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THE RED-SPOT DISEASE OF BROAD BEANS (VICIA FABA L.) CAUSED BY BOTRYTIS FABAE SARDIÑA IN CHINA[1]

T. F. YU

(Accepted for publication July 1, 1945)

INTRODUCTION

A leaf-spot disease of broad bean (*Vicia faba* L.) was noticed early in 1925 by Dr. R. H. Porter, who was, then, the plant pathologist of the University of Nanking, China. At his suggestion, investigations on the nature and cause of the disease and possible remedial measures were begun in the spring of 1927. Two years after this work was started, Sardiña (20) published on a leaf-spot disease of broad bean in Spain and attributed it to a new species, *Botrytis fabae*. The disease found in China is identical with that reported by Sardiña in Spain.

The work described in this paper was done from 1927 to 1929 and again from 1934 to 1937 in the Plant Pathology Laboratory, the University of Nanking, Nanking, China. The war stopped the work in November, 1937, and no opportunity of continuing the investigation or of publishing the results has occurred until now.

The name red spot was adopted in order to distinguish this disease from the chocolate spot of the same host which, according to Wilson (24) in England, is caused by *Botrytis cinerea* Pers.

HISTORY OF THE DISEASE

The red-spot disease has undoubtedly been present in many of the bean-growing regions for a long time. Early records of the chocolate-spot disease of broad bean attributed that disease to *Bacillus lathyri* Ma. and Taub. (14, 15, 17, 18, 19), but it has lately been demonstrated that *Botrytis cinerea* Pers. (25) and also *B. fabae* Sardiña (5, 8, 11) may cause chocolate spot.

The first authentic report of the red-spot disease was that by Sardiña (20) in 1929, in which the causal fungus was described as a new species of *Botrytis* (*Botrytis fabae* Sardiña). His second report appeared in 1931 (21) and two diseases of broad bean were described, one of which was attributed to *B. fabae* Sardiña, the other to a form of *B. cinerea* Pers. In these two papers the symptoms and etiology of the red-spot disease and the physiology and cultural characters of the pathogen were fully described.

In 1933, Ikata (8), probably without the knowledge of Sardiña's work reported from Japan the existence of a red-spot disease of broad bean caused by a new species of *Botrytis* which has also been named *B. fabae* n. sp. He described, in detail, the morphological and cultural characters of the pathogen as well as the control measures with fungicides.

[1] Paper No. 66, Plant Pathology Laboratory, the University of Nanking, Chengtu, China.

Other reports pertaining to *Botrytis* disease of beans were later given by Nattrass (11, 12, 13), Montemartini (10), Berger (3), El-Helaly (5), and Chorin (4).

DISTRIBUTION AND ECONOMIC IMPORTANCE

Red-spot disease of broad bean caused by *Botrytis fabae* Sardiña is widely distributed in the bean-growing regions throughout the world. It has been reported from Spain (20), Japan (8), England (24), Cyprus (11), French Morocco (3), Italy (10), Palestine (4), and Burma (17).

In China, it was first recorded by Porter in 1925 (see Annual report for the year 1924–25, College of Agriculture and Forestry, The University of Nanking, Nanking, China, 1926). He gave a brief report of its occurrence in Southeastern China and referred to it as "red spot" of broad bean. Surveys made by the writer from 1934 to 1937 inclusive revealed the wide distribution of the disease, especially along the Yangtze Valley and near the sea coast where it was observed in almost every field. In the inland regions, however, it was much less prevalent, while in the southwestern corner it was rare. Diseased specimens were also received from as far north as Hopeh province. In general, the disease seems to be co-existent with the crop throughout the country.

No exact figures are available regarding the amount of damage from red-spot disease of beans. The severity of the disease is correlated with weather conditions. In dry regions such as Yunnan of southwestern China, where the average monthly precipitation during March and April seldom exceeds 45 mm., the disease appears only as a few small spots on the leaf blades throughout the season. Consequently, it is of no economic importance. On the other hand, in regions where the atmospheric humidity is high, heavy losses to the crop result. The most severe outbreaks were observed along the river and near the sea coast, and there the disease is not only very prevalent but also destructive. Severe defoliation of bean plants and blight in the stems were seen in some of the worst cases. No experimental work had been done in determining the cause of stem blight, but *Botrytis cinerea* Pers. and *Bacillus fabae* were known to be capable of inducing blight in stems (26). The bacterial pathogen, a wound parasite, is very active in emphasizing blight and rot in stems under favorable conditions.

SYMPTOMS OF THE DISEASE

Red-spot disease of broad bean is most common on the leaves during the spring. The first signs of the disease are minute red spots on the leaf blade. As they enlarge, the center becomes depressed and the color changes to a chocolate or iron red with deep-colored margin. The spots are round, oval, or sometimes oblong and are generally numerous on a single leaf. Most of the spots are less than 1 mm. in diameter and usually remain small throughout the season. Their size probably is determined by the number per unit area. Spots as large as 4 or 5 mm. are also present. Isolations made from the large zonate spot yielded a typical culture of *Botrytis fabae*

Sardiña, and upon reinoculation with its spores on broad bean the characteristic small red spots were produced almost exclusively. Two or more spots may coalesce to form larger irregular lesions. The tip and margin of the leaf blade become black and papery and die. The dead tissues are easily cracked or torn, while the remainder of the leaf, although it bears numerous spots, may be a normal green for a time. Under moist conditions, the dead areas are soon covered with a light gray coating of conidiophores and conidia. With continued moist weather, there is still further development of the disease. The entire leaf blade may be involved and may collapse, although it remains attached. Excess moisture in the field results in the rot of the leaf. It is also not uncommon to find the plants defoliated, especially in a thickly sown field. Infection occurs mostly on the upper leaf surface. Spots on the lower leaf surface are few and large and have an indistinct margin. Long red stripes are also seen on the lower leaf surface. Microscopic examination of the affected leaf tissues shows that the mycelium of the fungus is intercellular.

Lesions on petiole and stem are oblong, elliptical, and deeply sunken at the center with a deep red margin. Their size varies considerably depending on the size of the affected plant part. On large stems, lesions may be 1 cm. long.

On pods, the fungus occasionally produces very tiny red spots which remain small throughout the season. No apparent damage from the fungus has ever been observed on the pods.

ISOLATION OF THE FUNGUS

The fungus is very easy to isolate. When any surface-disinfected portion of the diseased plant bearing the spots is placed upon a poured plate of potato or corn meal agar at ordinary room temperature, the fungus will develop rapidly. At first the colony consists of dirty white mycelial growth only, but after a few days spherical white bodies, which soon turn shiny black, appear at the center of the colony, and finally a large number of them form over its surface.

THE CAUSAL FUNGUS

Red-spot disease of broad bean in China is caused by *Botrytis fabae* Sardiña.

The hyphae are septate, branched, and usually coarse, both in lesions and in cultures. Dark-colored appressoria are commonly formed in culture by the branched hyphae which come in contact with glass containers.

Sclerotia are commonly found in cultures but so far have not been observed upon the host in nature. Newly formed sclerotia in cultures appear as white masses of mycelium. As development continues, the surface becomes waxy and light olivaceous green, then gradually darkens, until a hard black mass results. Mature sclerotia are round, oblong, or irregular. When disinfected diseased leaves are incubated in moist Petri dishes, round or very slender sclerotia are formed in great abundance along the leaf vein.

On ordinary culture media at room temperature, sclerotia are 0.5–3 × 0.6–3.5 mm. with an average of 1 × 1.5 mm. The size of sclerotia is greatly influenced by temperature and by composition of the substratum. Sclerotia may be in rings or scattered on the surface of agar in dishes. It is not uncommon to find them aggregated into crusty masses. They germinate either by sending out mycelium or by producing conidiophores and conidia upon their surfaces. Direct germination of sclerotia, however, has been less common throughout the present investigation.

Conidia may be produced directly from fine mycelium or from sclerotia. They are septate, twisted, and from 8 to 20 μ in diameter and 600 to 1500 μ in length. The growing tip of the conidiophore sends out several side branches from the main stem and these, in turn, branch. The ends of these branches swell and send out the sterigmata on which lateral conidia are produced. Conidia are produced profusely in clusters that are either terminal or intercalary.

Conidia are subglobose to oval, hyaline, and 12.2–22.8 × 10.5–15.8 μ with an average of 16.7 × 13.7 μ. Under favorable conditions they germinate in a few hours by sending out one or two germ tubes from each end.

Microconidia are abundant on culture media, especially media poor in carbohydrate. They are hyaline, globose, or oval and 2.9–4 μ in diameter with an average of 3.6 μ. No germination of microconidia has ever been observed. Considerable variation is noticeable in the type of conidiophore. The most frequent form is a cluster of conidiophores arising directly on a cell of the sterile hyphae. In another type a special branch arises from the sterile hyphae. In still another form a long slender branch bears upon its tip either a single conidiophore or a group of conidiophores. Microconidia originate from at least three sources, mycelium, germinating conidia, and germinating sclerotia. It is interesting to note that sclerotia, when placed on an agar surface after a certain period of desiccation, will germinate by producing an extremely slow growing mycelium. Small white and green mycelium-like tufts are scattered over the agar surface. These are the clusters of microconidia.

PHYSIOLOGY OF THE CAUSAL FUNGUS

Cultural Studies

In studying the cultural characters of *Botrytis fabae,* both natural and artificial culture media were used. No attempt is made to give detailed descriptions of the growth characters of the fungus on each kind of medium. In general, it thrives well on all the media commonly used in the laboratory. It grows rapidly and forms a fluffy mycelium which later becomes appressed to the surface of the agar and has a glistening appearance, and sclerotia then begin to form. Aerial mycelium is scant and is usually at the top of an agar slant. Sclerotia and microconidia are usually present. Conidia, although formed abundantly in nature, are produced only occasionally in artificial media in the laboratory. It has been pointed out repeatedly by

the previous investigators that the production of conidia is influenced by the amount of moisture in the air (7, 9, 25). Attempts were made to induce production of conidia, but the partial drying of plate cultures described by Hopkins (7) produced only inconsistent results. Undoubtedly the production of conidia is largely a matter of moisture relations, yet the possible influence of other unknown factors must also be considered.

Temperature in Relation to Growth of Mycelium

The relation of temperature to the rate of mycelial growth on plates of potato-dextrose agar was studied. Separate dishes were inoculated with the fungus and placed in constant temperature chambers, adjusted to temperatures between 5° and 36° C. Two experiments were made. In the first experiment, abundant mycelial growth occurred between 20° and 27° C. within four days. In the second experiment, cultures incubated at 24° to 26° C. grew better than those at 15°–17° C. or at 30° C. in the same length of time. The optimum temperature for mycelial growth of the fungus lies somewhere between 24° and 26° C. (Table 1). Cultures incubated at 5° to 7°

TABLE 1.—*The relation of temperature to growth of mycelium of Botrytis fabae Sardiña*

Experiment 1 (4 days)		Experiment 2 (4 days)	
Temperature, in degrees C.	Diameter of the colony[a]	Temperature, in degrees C.	Diameter of the colony[a]
	cm.		*cm.*
19–19.6	3.8	15–17	2.3
20–20.4	5.3	24–26	5.6
21–22	6.7	30–34	2.1
25–25.3	6.9		
27–28	6.1		

[a] Average of ten plate cultures.

C. produced slight growth by the eighth day, and on the 17th day the colonies had attained diameters from 0.5 to 2 cm. and sclerotia were appearing on the surface. At 36° C., very little growth was seen for a long time. The minimum temperature for mycelial growth, therefore, probably is about 6° C. and the maximum about 36° C.

Relation of pH to Growth of Mycelium

The effect of pH of culture medium on the rate of mycelial growth was determined from the dry weight of the mycelia produced in the standard nutrient solution incubated at 24° C. for seven days. Determination of the pH was by both the colorimetric and electrometric methods. The average dry weight of the mycelium, including few small sclerotia, of ten flasks for pH 3.2, 4.4, 5.2, 6.2, 8.1, and 8.5 were respectively 0.00, 0.25, 0.26, 0.14, 0.13, 0.12, and 0.00 mgm. The optimum pH for mycelial growth lies between 4.4 and 5.2. El-Helaly (5) found that the fungus thrives best at pH 4.5,

while Sardiña (21) found the optima for mycelial growth of two strains of the fungus at pH 5.3 and at pH 7.3.

Germination of Conidia

Germination of conidia of *Botrytis fabae* was tested at various temperatures. A spore suspension was made in tap water and drops were transferred to clean glass slides placed in moist Petri dishes in the incubators. In the first test the conidia germinated best between 20° and 25° C. within 24 hours (Table 2). If the duration of the test was prolonged, the conidia germinated over a wide range of temperatures. In a second test, attempts

TABLE 2.—*Relation of temperature to germination of conidia of Botrytis fabae Sardiña*

Experiment 1 (24 hours)		Experiment 2 (48 hours)	
Temperature, in degrees C.	Percentage of germinating conidia	Temperature, in degrees C.	Percentage of germinating conidia
10–11	0.0	15–17	76.8
14–15	11.1	17–19	85.4
20–21	68.9	19–21	93.0
24–25	58.9	22–23	74.1
29–30	23.6		

were made to determine more exactly where the optimum temperature lies. It is between 19° and 21° C. This optimum is close to that found by Ikata (8) who reported it at about 20° C. The maximum and minimum temperatures for conidium germination were 34° and 5° C., respectively.

HOST RANGE OF THE FUNGUS

In inoculation tests, Sardiña (21) found that *Botrytis fabae* is unable to infect plants outside the Leguminoseae. Among the leguminous plants, he obtained infection on French bean. Ikata (8) inoculated *Vicia faba, Pisum sativum, Phaseolus vulgaris, Astragalus sinensis, Medicago sativa,* and a second species of *Vicia* with a suspension of conidia of the fungus and found none of these plants, except its own host (*V. faba*), was infected. Schnellhardt and Heald (22), in connection with their studies on the decays of apple, made artificial inoculation on the fruit with various species of *Botrytis* including *B. fabae* Sardiña. This fungus was only weakly parasitic to apples. Nattrass (12) observed a chocolate-spot disease of *Vicia sativa* in nature. The sporulation of its causal fungus closely resembled that of *B. fabae* Sardiña and when broad beans were artificially inoculated with a suspension of conidia, the characteristic chocolate spots were produced.

In the course of the present investigation, inoculation experiments were repeatedly made for determining the host range of the fungus. The results obtained indicate that *Botrytis fabae* will not attack non-leguminous plants such as potato, tomato, pepper, eggplant, lettuce, carrot, celery, radish, and cabbage. Inoculations with suspensions of conidia on the following

leguminous plants also gave negative results: *Arachis hypogaea* L., *Cercis chinensis* Bunge, *Dolichos lablab* L., *Glycine max* Merr., *Indigofera spp.*, *Lespedeza bicolor* Turcz., *L. striata* Hook. & Arn., *Lupinus albus* L., *Medicago sativa* L., *Melilotus alba* Desr., *M. officinalis* (L.) Lam., *Phaseolus angularis* Wight, *P. aureus* Roxb., *P. mungo* L., *P. vulgaris* L., *Robinia pseudoacacia* L., *Sophora japonica* L., *Trifolium incarnatum* L., *T. pratense* L., *T. repens* L., *Vicia tetrasperma* (L.) Moench., *V. villosa* Roth., *Vigna sinensis* Savi., *Wisteria sinensis* Sweet. The leguminous plants that were infected were *Vicia faba* L., *Pisum sativum* L., *P. sativum* var. *arvense* Poir., and *Vicia sativa* L. The fungus invariably produced small red spots on *Vicia sativa* L. when it was artificially inoculated. In the case of *Pisum sativum* L. and *P. sativum* var. *arvense* Poir., the results obtained were variable. In a few tests, the fungus failed to infect the plants.

OVERWINTERING EXPERIMENTS

Experiments were undertaken to determine by what means the fungus lives through the winter. Sclerotia produced on sterile soybean stems in flasks were placed in a wire basket and left in a bean field where they were exposed to prevailing weather at Nanking from November to March in the winters of 1928, 1934, and 1935. Sclerotia were viable at the end of the winter in all three years.

In another experiment, sclerotia taken from potato-dextrose-agar plates were either left on the soil surface in the field or buried about two cm. below the soil level in pots containing fine clay soil. The experiment was started Nov. 1, 1934. Forty days later, abundant sporulation was seen on the surface of sclerotia which had been left on the soil surface. None of the buried sclerotia had germinated at the end of 68 days. They were then turned and brought to the soil surface, and there was abundant sporulation about a month later. These experiments indicated conclusively that sclerotia may carry the disease through the winter.

In order to find out whether or not conidia might live over the winter, experiments of the viability of conidia under desiccation were made. Drops of spore suspension made of fresh spores were placed on clean glass slides in a dry container hanging on a tree in the field and left under drying conditions. Germination tests were made at regular intervals until no germination was observed. About 80 per cent of the spores germinated at the end of 70 days. After this, the percentage of spore germination dropped sharply to about twenty. At the end of 140 days, few spores still retained their viability. Since the broad bean crop is harvested in the middle of June and the disease has never been observed in November under natural conditions, it is improbable that conidia produced on the affected plants of the first year cause infection in the spring of the next year.

LIFE HISTORY STUDIES

It has been stated above that sclerotia have never been observed on the diseased plants under natural conditions. However, when affected stems

were cut and piled in a low moist shady place in a field, sclerotia were occasionally found either on the surface or underneath the epidermis of the stems during July or August. In this connection, it is interesting to mention that in certain inoculation experiments, sclerotia had been found underneath the epidermis of the inoculated plants that were kept under moist conditions for a considerable time. The writer is inclined to believe that the production of sclerotia under natural conditions, if it occurs, is determined by proper moisture and temperature conditions as well as by the stage of maturity of the host plants.

In the overwintering experiments, the fungus survived the dormant period as sclerotia. Sclerotia germinate in the spring and produce abundant conidia which bring about primary infection. This primary infection is tremendous because sclerotia may produce several crops of spores in a short time. In one laboratory experiment, sclerotia with conidia taken in from the fields on March 22 were washed thoroughly with tap water and incubated in moist dishes. Conidia were again formed in less than 24 hours. The sclerotia were washed again, dried, and again washed. The procedure has been repeated as many as six times in 24 days and conidia were still produced by the sclerotia.

Under favorable conditions, conidia produced in great abundance on the first-infected leaves furnish abundant inoculum for the second infection, which occurs commonly in May. A great number of spores were caught on dishes containing agar or on slides coated with vaselin when these devices were exposed in a bean field. The prevailing high atmospheric humidity is extremely favorable for production of conidia and for infection by the fungus.

INFLUENCE OF CLIMATIC CONDITIONS

Red-spot disease of broad bean has been found in all the bean-growing districts along the Yangtze river and near the sea coast of China, but it is rare in many other regions of the country. Diseases caused by *Botrytis* are generally prevalent and severe under high moisture (1, 2, 6, 16, 23, 25). In case of the present disease, Sardiña (21) found that a relative humidity of at least 85 per cent is necessary to induce infection on beans. Experiments by the writer also indicated that a relative humidity of 90 per cent or above is essential for infection as well as for production of conidia on affected plant parts.

The influence of moisture on this disease may be better understood by comparing meteorologic data, for the two months prior to the harvest of the crop in a dry and a wet region. They may be represented by Kunming in southwest China and Nanking along the river. In Kunming, the disease appears rarely, whereas in Nanking it occurs in great abundance. The average monthly precipitation in Kunming for March and April in the five years from 1928 to 1933 were respectively 38 and 47 mm. Relative humidities for the same months were respectively 60.5 and 61.8 per cent, amounts

far below the minimum as shown both by Sardiña (21) and the writer. In Nanking, the average monthly precipitation for April, May, and June in 1933 was respectively, 114.1, 116.4, and 135 mm., and the relative humidity for the same months was respectively 83.2, 85.2, and 93.1 per cent. The difference in moisture conditions apparently explains the incidence of the disease in these two regions.

SUMMARY

A leaf-spot disease of broad bean in China, here referred to as red-spot disease, is found to be identical with that described by Sardiña in Spain and is caused by *Botrytis fabae* Sardiña. The disease is widely distributed in China, especially in the bean-growing regions along the Yangtze river and near the sea coast. Under suitable environmental conditions, affected leaves may collapse or plants may be so defoliated that pods do not form.

The symptoms of the disease and the morphology and physiology of the causal fungus have been described.

The fungus appears to be limited to a few species of the Leguminoseae. Of a large number of leguminous plants exposed to artificial inoculation, only *Vicia sativa* L., *Pisum sativum* L., and *P. sativum* var. *arvense* Poir., in addition to *Vicia faba* L., were slightly infected. The fungus so far has been unable to attack plants outside the Leguminoseae.

Overwintering experiments indicate that the fungus may overwinter by means of sclerotia, although they have never been observed on diseased plants in nature. The disease is probably not carried through the winter by conidia produced on the affected plants of the previous season.

The prevalence of red-spot disease in the various bean-growing regions is correlated with rainfall and atmospheric humidity. The scarcity of the disease in the southwest is correlated with the dry climate whereas its common occurrence along the river or near the sea coast is correlated with high atmospheric moisture during the two months prior to the harvest of the beans.

No attempts have been made to control the disease.

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DISTRIBUTION OF RACES OF TILLETIA CARIES AND TILLETIA FOETIDA AND THEIR RELATIVE VIRULENCE ON CERTAIN VARIETIES AND SELECTIONS OF WHEAT[1]

H. A. Rodenhiser and C. S. Holton[2]

(Accepted for publication July 10, 1945)

INTRODUCTION

Experiments were started in 1932 to obtain information on the number, distribution, and prevalence of physiologic races of *Tilletia caries* (D. C.) Tul. (*T. tritici* (Bjerk.) Wint.) and *T. foetida* (Wallr.) Liro (*T. levis* Kuehn) in the different wheat-growing regions of the United States. In previous papers (4, 7) 14 races of *T. caries* and 10 of *T. foetida* were described on the basis of the reaction of certain differential varieties of winter and spring wheats. These data, together with information on the prevalence and distribution of the races, are basic to a well-rounded program for the development of bunt-resistant varieties. Data are presented here (1) on the identification of 7 previously undescribed races, (2) on the distribution of species and races in the principal wheat-growing areas of the United States, and to a limited extent in Mexico, and (3) on the relative virulence of races on certain winter and spring wheat varieties and hybrids that may be of interest to wheat breeders in connection with the development of bunt-resistant varieties.

NEW PHYSIOLOGIC RACES

The methods used in the identification of bunt races have already been described (4, 7). The reactions of differential wheat varieties to all known races, including 2 new races of *Tilletia caries* (T–15 and T–16) and 5 of *T. foetida* (L–11 to L–15, inclusive) are in table 1. The original source of each of the 7 new races and the distinguishing pathogenic characters of each are shown in table 2. Among the races previously described, there are several that differ from each other in relative degrees of virulence on the same differential hosts. This is likewise true of 6 of the new races here described and their occurrence probably does not change greatly the factors to be considered in breeding for bunt resistance. However, the new races T–15 and T–16 complicate the breeding program somewhat since one may no longer expect to obtain varieties from combinations of Hohenheimer, Hussar, and Oro that are resistant to all known races. It should be possible, however, to obtain the desired resistance by bringing in the so-called Ridit factors which control resistance to both of these races.

[1] Cooperative investigation of the Division of Cereal Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture, and the Agricultural Experiment Stations of Washington and Idaho.

[2] Senior Pathologist and Pathologist, respectively, Division of Cereal Crops and Diseases.

TABLE 1.—*Reactions*[a] *of differential wheat varieties to physiologic races of Tilletia caries and T. foetida*

Species and race	Hybrid 128	Ridit	Oro	Hohenheimer	Hussar	Albit	Martin	White Odessa	Ulka	Marquis	Canus
T. caries											
T–1	S	R	R	R	R	R	R	R	S	I	R
T–2	S	R	R	R	R	R	R	R	S	R	R
T–3	S	R	R	R	R	R	R	R	S	S	S
T–4	S	R	R	R	R	I	S	S	S	S	R
T–5	S	R	R	R	R	I	S	S	S	S	S
T–6	S	R	R	R	R	S	S	S	S	S	R
T–7	S	R	R	R	I	S	S	S	S	S	I
T–8	S	R	R	R	S	S	S	S	S	S	S
T–9	S	R	R	I	R	R	R	R	S	I	R
T–10	S	R	R	S	R	R	R	R	R	I	R
T–11	S	S	R	R	R	R	R	R	I	S	S
T–12	S	R	R	S	R	S	I	S	S	R	R
T–13	S	S	R	R	S	S	R	S	I	S	I
T–14	S	R	R	R	R	S	R	S	S	R	R
T–15	S	R	R	S	S	S	S	S	S	S	S
T–16	S	R	S	S	R	R	R	R	S	I	I
T. foetida											
L–1	S	R	R	R	R	R	R	R	S	I	R
L–2	S	R	R	R	R	R	R	R	S	S	R
L–3	S	R	R	R	R	R	R	R	S	S	S
L–4	S	R	R	R	R	S	S	S	S	I	R
L–5	S	R	R	R	R	S	S	S	S	S	S
L–6	S	R	R	R	I	S	S	S	S	S	S
L–7	S	R	R	R	S	S	S	S	S	I	S
L–8	S	R	S	R	R	R	R	R	S	S	S
L–9	S	S	R	R	I	S	R	S	S	I	S
L–10	S	I	R	R	R	R	R	R	S	I	S
L–11	S	R	R	R	R	R	R	R	S	I	I
L–12	S	R	R	R	R	S	R	R	S	I	R
L–13	S	R	R	S	R	R	R	R	S	S	R
L–14	S	R	R	R	R	S	I	S	S	S	R
L–15	S	R	R	R	S	S	S	S	S	S	I

[a] R = Resistant (0–10 per cent infection); I = Intermediate (11–40 per cent infection); S = Susceptible (41–100 per cent infection).

Races L–13, L–14, and L–15 were identified from the collections made in Mexico (Table 2). L–13 is the only race of *Tilletia foetida* that is pathogenic on the variety Hohenheimer. In this respect it is similar to T–10, but the two differ in pathogenicity on Ulka and Marquis. Races L–14 and L–15 have the same host range as L–4 and L–7, respectively. The distinction between L–14 and L–4 is based on the degree of virulence on Martin and Marquis, while L–15 and L–7 differ in degree of virulence on Marquis and Canus.

DISTRIBUTION OF SPECIES AND RACES

Species

Surveys to determine the distribution of *Tilletia caries* and *T. foetida* have been made by several investigators (2, 6, 13). It is evident from their

reports that *T. caries* occurs over a considerable area of the United States but its prevalence is most marked in certain regions. It is common in the durum wheats grown in the Upper Mississippi Valley and greatly increases in relative prevalence toward the intermountain area of the Northwest where it has been the predominating species. Elsewhere in the United States *T. foetida* has always predominated and in recent years it has increased in relative prevalence in the Northwest. In fact, according to Kienholtz and Heald (6), only *T. caries* was known to occur in the State of Washington prior to

TABLE 2.—*Original source and differential characteristics of seven new races of Tilletia caries and T. foetida*

Species and race	Collection No.	Source and differential characteristics
T. caries		
T–15	571	From Oro wheat in 1938 near Cottonwood, Idaho, collected by W. M. Bever, who made preliminary tests on differential hosts and reselected it from Hohenheimer at Moscow, Idaho. It is similar to T–12 on several varieties but differs from this race by the susceptibility of Hussar, Martin, Marquis, and Canus.
T–16	346	From a field of Leap wheat in 1938 near Charlestown, W. Va. It is characterized by the susceptibility of Oro and Hohenheimer and the intermediate reaction of Marquis and Canus. It is the only race that is pathogenic on both Oro and Hohenheimer.
T. foetida		
L–11	264	From a field of mixed wheat in 1935 near Sutter, California. It is similar to race L–1 and differentiated from it only by the reaction of Canus which is resistant to L–1 and intermediate to L–11.
L–12	278	From a field of Poole Wheat in 1936 near Staunton, Va. It is characterized by the susceptibility of Albit and the resistance of all of the other winter wheat differential varieties.
L–13	308	From a field of mixed wheat in 1936 near Mexico City, Mexico. It is characterized by the susceptibility of Hohenheimer. This is the only known race of the species to which this variety is susceptible.
L–14	307	From a field of mixed wheat in 1936, 30 miles northwest of Mexico City, Mexico. It is similar to race L–4 but differentiated from it by the intermediate reaction of Martin and the susceptible reaction of Marquis.
L–15	311	From a field of mixed wheat in 1936 near San Juaquin, Mexico. This race is similar to L–7 but is differentiated from it by the susceptibility of Marquis and intermediate reaction of Canus.

1918 but surveys made in 1927 and 1928 showed that *T. foetida* was present in all the principal wheat-growing districts of this State. However, *T. caries* was still the predominant species. In 1929 and 1930 Flor (2) made collections of bunt from 182 fields in the principal wheat-producing areas of Oregon, Washington, and northern Idaho, and found *T. caries* in every collection and *T. foetida* in 60 per cent of the collections.

The present report is based on studies with 369 collections[3] of *Tilletia caries* and *T. foetida* made from 1932 to 1942. As shown in tables 3 and 4, they were obtained from 35 states in the United States, 6 states in Mexico,

[3] A few field collections were mixtures of the two species or more than one race. When isolated each was given a separate collection number.

and 2 provinces in Canada. Of the total number of collections 62 were *T. caries,* obtained from 15 states and provinces, and 307 were *T. foetida,* obtained from 41 states and provinces. Thus, *T. foetida* has a much wider distribution than *T. caries,* the former being found in all except one of the States and Provinces from which collections were obtained and the latter in less than a third of the collections. The increase of *T. foetida* in the Northwest noted in previous surveys is corroborated by the results of the present survey. Of 94 collections made in the far western states, including

TABLE 3.—*Number and distribution of physiologic races of Tilletia caries in 62 collections from North America.*

Source of collections	Number of collections of races																Total collections	Number of races
	T-1	T-2	T-3	T-4	T-5	T-6	T-7	T-8	T-9	T-10	T-11	T-12	T-13	T-14	T-15	T-16		
United States																		
Arizona	2																2	1
California	15			1													16	2
Georgia		1															1	1
Idaho	1					1								3	1		6	4
Minnesota		1															1	1
Montana			1			1	1	2			1						6	5
New York				1	1												2	2
North Dakota		4															4	1
Oregon			1			1		1	1			1					5	5
South Dakota		1															1	1
Washington	1					1	3	1	1	1	1		1				10	8
West Virginia								1								1	2	2
Wyoming		2															2	1
Canada																		
Manitoba						1											1	1
Mexico																		
Sonora	2		1														3	2
Totals	21	9	3	2	1	5	4	5	2	1	2	1	1	3	1	1	62	
Per cent	33.9	14.5	4.8	3.2	1.6	8.1	6.5	8.1	3.2	1.6	3.2	1.6	1.6	4.8	1.6	1.6		

Colorado and Wyoming, 41 were *T. caries* and 53 were *T. foetida.* East of the Rocky Mountains, *T. foetida* continued to predominate as shown by the fact that, of 238 collections, 221 were *T. foetida* and only 17 were *T. caries.* It seems evident that *T. foetida* has spread to new regions more readily than *T. caries.* The explanation for this apparent wider adaptation of *T. foetida* is not indicated by the data at hand.

Races of Tilletia caries

The distribution of the races of *Tilletia caries* is shown in table 3. The 16 races of this species, identified in 62 collections, were distributed in 13 states in the United States, one state in Mexico, and one province in Canada.

Thus, on the average, a different race was identified for approximately every four collections of the species. This high ratio is partly explained by the fact that several races that were previously identified by other investigators

TABLE 4.—*Number and distribution of physiologic races of Tilletia foetida in 307 collections from North America*

Source of collections	Number of collections of races															Total collections	Number of races
	I–1	I–2	I–3	I–4	I–5	I–6	I–7	I–8	I–9	I–10	I–11	I–12	I–13	I–14	I–15		
United States																	
Arizona	2															2	1
California	2	4	2					1			3					12	5
Colorado			1	1				5								7	3
Delaware	1										1					2	2
Georgia	3															3	1
Idaho			1				1	1	1	1						5	5
Illinois	4		3	4						1						12	4
Indiana	5	1		5												11	3
Iowa			3													3	1
Kansas			6		2		2									10	3
Kentucky	2				1						1	1				5	4
Maryland	2			1												3	2
Michigan	6			3								1				10	3
Minnesota	1	4														5	2
Missouri	2		4	5							1	1				13	5
Montana		6	11	1		1		2							1	22	6
Nebraska		4	3				2									9	3
Nevada	1															1	1
New York	6			2							1					9	3
North Carolina	8			3												11	2
North Dakota		15	1													16	2
Ohio	6		1	4							2					13	4
Oklahoma			10	1						7						18	3
Oregon	1		1	1			1									4	4
Pennsylvania	3			1												4	2
South Carolina	2															2	1
South Dakota		9	2	1												12	3
Tennessee	5			1								1				7	3
Texas			3							3	1					7	3
Utah			1	1			3	1								6	4
Virginia	8			1							3	1				13	4
Washington	2		3				2			1						8	4
Wisconsin				1												1	1
Wyoming	1	3	4							1						9	4
Canada																	
Alberta		3														3	1
Mexico																	
Coahuilla	2	7												2		11	3
Jalisco		1												3		4	2
Mexico													1	2	1	4	3
Michoacán	2															2	1
Puebla		3						1							2	6	3
Sonora	1	1														2	2
Total number	78	61	60	37	3	1	11	11	1	14	13	5	1	7	4	307	
Per cent	25.4	19.9	19.5	12.1	1.0	0.3	3.6	3.6	0.3	4.6	4.2	1.6	0.3	2.3	1.3		

were included in the collections. Therefore, strictly speaking, all of the so-called collections were not actually field collections.

The number of collections of each race ranged from 1 to 21. Races T–1 and T–2 were the most prevalent, constituting 33.9 and 14.5 per cent, respectively, of the collections of *Tilletia caries*. The former, identified only from collections in four western states and Sonora, Mexico, greatly predominated in collections from California where 15 of the 16 collections of this species were T–1. That it was collected only once in Idaho and once in Washington probably is due to the fact that these states had been thoroughly surveyed by earlier workers and in the present studies emphasis was placed on collections from varieties that are resistant to T–1. Race T–2 was identified in collections from five states over the hard red spring and the hard and soft red winter wheat areas but it is apparently commonest in the durum section of the hard red spring wheat area. As shown in table 1, this race is relatively innocuous on all but one of the winter wheat differential varieties; however, it is highly virulent on Mindum and certain other durums, thus complicating the problem of breeding for bunt resistance in this class of wheats. Races T–6 and T–8 were found in 5 collections each, the former being obtained from 5 areas and those of the latter from 4 areas. Both of these races are important factors in the winter and spring wheat improvement program. Six races, namely, T–5, T–10, T–12, T–13, T–15, and T–16, were each found in only one collection. However, this does not greatly minimize their importance, because of the fact that bunt is accumulative. Consequently, in a wheat improvement program, all races should be considered, even though for the time being some of them appear to be of uncommon occurrence.

Classification of the dwarf bunt fungus in relation to the races of *Tilletia caries* that cause ordinary bunt is being held in abeyance until methods have been devised whereby comparable pathogenicity tests may be made. However, because of the importance of dwarf bunt in certain wheat-growing areas, information on its occurrence and distribution is needed. Dwarf bunt first attracted attention as a regional problem in wheat production in 1931. At that time it was observed[4] in devastating proportions over a fairly wide area in southeastern Idaho and northern Utah. In the same year it was observed on the High Prairie area of Klickitat County in south-central Washington by Roderick Sprague and in the Gallatin Valley near Bozeman, Montana, by P. A. Young and H. E. Morris. Since then, other reports of dwarf bunt have shown that it now occurs over a much wider area in these states.

The most extensive area of dwarf bunt infestation apparently is in Idaho where it occurs (1) throughout the dry-land wheat area of the southeast portion; (2) in the prairie region around Winchester and Nesperce; (3) on the ridges east of Troy; (4) in the Palouse near Genessee; and (5) in the semi-timbered section around Worley and Plummer. Thus, in Idaho, the

[4] Observations reported by representatives of the Division of Cereal Crops and Diseases and cooperating agricultural experiment stations in Idaho and Utah.

infested areas are distributed from the southern border to the panhandle. In Washington, in addition to the infested area on High Prairie (Klickitat County), which extends eastward to the Goldendale area, dwarf bunt has been observed near Waterville (Douglas County), near Wilbur (Lincoln County), and near Spangle (Spokane County). The occurrence of dwarf bunt in Wyoming, adjacent to the southeastern border of Idaho, has been reported by Blodgett (1).

Dwarf bunt infestation near Bozeman, Mont., was observed as early as 1931. Recently it has been reported by R. H. Bamberg and H. E. Morris to occur in destructive proportions in the northwestern part of the State extending from 20 miles south of Flathead Lake to 10 miles north of Kalispell. In Utah, the first area of infestation observed was in the Cache Valley near Logan, Cache County, and in Box Elder County by D. C. Tingey and R. W. Woodward. During the past 10 years it has spread and is now established over the entire dry farm areas of the northern part of the State. It also is locally distributed in Salt Lake County and as far south as the eastern end of Juab County near Nephi.

Dwarf bunt has been found also in Colorado[5] and New York[6] as indicated by reports verified by specimens examined by the writers.

Races of Tilletia foetida

A total of 307 collections, or 83.2 per cent of those studied, proved to be *Tilletia foetida*. The distribution of the 15 races identified is shown in table 4. They were distributed in 34 states in the United States, six states in Mexico, and one province in Canada. Thus, on the average a different race was identified for approximately every 20 collections. The number of collections of each race ranged from 1 to 78. Races L–1, L–2, L–3, and L–4 predominated with respect to number of times collected and range of distribution. Of these, L–1 was most prevalent and widely distributed, having been collected 78 times in a total of 25 states, including those of Mexico. Sixty-one collections from 13 states and provinces yielded L–2, and 60 collections from 18 states, in the United States only, yielded L–3. Next in relative prevalence was L–4, which was found in 37 collections from 18 states, in the United States only. L–2 and L–3, which have a limited range in pathogenicity on the winter wheat differential varieties, were the commonest races of *T. foetida* collected in the hard red spring wheat area. One of the five new races of *T. foetida*, L–11, reported for the first time in this paper, was widely distributed, having been collected in seven eastern and midwestern states and in California.

The twenty-nine collections of *Tilletia foetida* from 6 states in Mexico yielded 6 races, three of which are new, namely, L–13, L–14, and L–15. Of these three, only L–15 has been found in the United States. Race L–2, which is among the four most common races in the United States, also was the most

[5] Report by E. W. Bodine and L. W. Durrell in Plant Disease Reporter 25: 485, 1941; and in correspondence with E. F. Darley.

[6] Observed by B. B. Bayles who furnished specimens for laboratory study.

TABLE 5.—*Percentage of bunt produced on certain varieties and strains of Triticum aestivum L., T. durum Desf., and T. timopheevi Zhuk. by physiologic races of Tilletia caries and T. foetida*

Species, class, variety, or hybrid	C.I. number	Percentage of culms infected by *Tilletia caries* ("T" races)																Percentage of culms infected by *Tilletia foetida* ("L" races)								
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5	7	8	9	10
T. aestivum																										
Hard red spring																										
Hard Fed. × Hussar	11792	0	0	0	0	10	0	10	48	0	0	0	0	2	0	78	0	3	0	0	0	35	80	1	0	0
Henry	12265	56	4	0	19	51	9	69	49	61	45	15	61	31	26	51	37	45	56	35	36	30	3	48	4	13
Hope	8178	3	2	0	1	2	0	1	1	0	0	0	0	2	0	0	0	2	8	7	0	5	2	8	2	0
Komar × Hussar	11715	0	0	0	0	1	0	2	6	7	0	1	0	8	0	9	0	0	0	0	0	4	5	2	2	0
Merit	11870	42	2	6	2	31	20	24	45	29	8	16	23	7	3	10	9	78	68	26	28	16	15	22	2	20
Mida	12008	0	0	0	0	11	0	4	9	4	0	0	11	0	0	1	10	3	7	3	0	T	1	19	0	8
Newthatch	12318	15	6	20	30	39	22	32	39	21	24	29	9	24	7	20	9	34	37	44	39	29	23	22	29	30
Pilot	11945	17	4	3	5	21	17	27	46	2	1	7	5	19	0	1	3	20	67	40	28	34	4	44	5	22
Regent	12070	24	15	13	4	6	5	8	35	13	2	30	4	21	0	9	17	8	21	35	5	3	8	65	10	31
Regent × Pilot	12317	3	T	T	1	6	T	2	6	1	2	2	5	1	0	1	1	1	3	1	0	1	1	3	0	1
Reliance 1018 × Mercury	12204	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0
Renown	11947	1	0	0	0	0	1	0	2	0	0	0	6	0	0	0	0	2	6	2	0	1	0	1	0	0
Rival	11708	23	3	1	0	2	2	5	3	5	2	5	4	8	0	34	6	23	28	26	6	17	7	46	7	27
Timopheevi × Steinwiedal	12347	24	35	50	40	31	63	72	40	62	3	7	45	3	1	78	70	53	62	78	58	58	71	52	16	41
Thatcher	10003	42	20	40	32	61	56	46	52	6	14	16	2	16	6	12	12	71	69	72	72	73	28	35	24	28
Vesta	11712	45	15	27	46	73	74	72	22	13	1	1	3	1	0	23	97	54	79	79	64	58	49	63	12	70
Hard red winter																										
Cache	11599	0	1	2	1	0	1	0	5	5	2	34	1	27	1	3	8	0	0	0	0	T	0	2	16	12
Comanche	11673	1	3	7	0	2	1	2	2	2	2	1	1	2	1	3	36	14	1	2	2	1	5	86	2	2
H–44 × Minturki	12414	0	2	1	0	1	6	2	T	1	2	6	0	3	6	15	29	3	5	8	2	0	8	0	1	0
H–44 × Minturki	12022	0	0	0	0	0	0	2	2	0	0	1	0	1	2	2	35	1	0	0	2	1	1	57	0	0
Hope × Minturki	12415	10	2	30	8	14	9	16	34	4	8	16	7	16	11	20	36	10	15	24	19	13	31	18	18	16
Minturki	6155	7	0	22	7	11	3	7	3	1	4	17	1	80	15	66	0	0	7	17	9	11	27	64	71	0
Nebred	10094	T	0	1	1	T	1	2	1	2	0	1	0	3	0	10	27	.....	2	6	2	11	3	91	6	7
Oro × Turkey-Florence	11865	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	8	0	0	0	0	0	1	1	0	0
Pawnee	11669	8	42	13	38	3	44	26	46	30	21	37	11	54	10	36	35	19	13	15	12	20	52	41	20	24
Relief	10082	0	0	1	0	5	0	1	24	3	0	7	0	25	0	10	0	1	0	0	0	4	73	1	13	0
Rex × Oro	12421	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	2	0	0
Rex × Rio	12234	0	0	0	0	T	T	T	T	0	0	0	0	0	0	0	1	0	0	0	1	1	2	2	0	0
Ridit × 15a	11924	2	0	2	1	9	2	2	9	16	1	53	2	51	4	18	16	0	1	1	1	2	13	1	29	17
Rio	10061	T	1	1	0	T	1	1	4	0	0	0	2	0	0	0	54	1	0	1	T	0	T	97	0	0
Rio × Rex	12422	0	0	0	4	1	3	1	0	0	0	0	0	0	0	1	0	0	0	0	0	1	4	1	0	0
Turkey	11530	0	0	0	4	12	6	9	26	0	0	0	11	37	10	41	0	0	0	0	17	11	60	0	34	0
Wasatch	11925	0	0	0	0	0	0	5	13	7	0	9	0	37	T	0	0	0	0	0	0	0	1	0	30	0
Yogo	8033	1	1	2	3	9	1	13	3	1	2	3	2	8	2	12	46	7	3	7	3	2	11	90	7	20

Species, class, variety, or hybrid	C.I. number	Percentage of culms infected by *Tilletia caries* ("T" races)																Percentage of culms infected by *Tilletia foetida* ("L" races)								
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5	7	8	9	10
Soft red winter																										
Fultz × Hungarian	12017	1	0	0	18	78	11	86	83	2	0	13	18	48	24	.....	.....	1	4	0	21	32	83	7	29	1
Hope × Hussar	11682	1	9	27	5	18	0	3	17	11	T	24	2	38	1	35	19	1	5	47	3	10	35	41	29	24
Hus. × Hohenheimer	10068–1	0	0	0	0	0	0	0	0	1	2	0	T	4	1	80	24	0	0	0	0	0	0	0	1	0
Hus. × Red Wave	12423	0	0	0	2	38	0	72	70	0	0	18	3	55	4	.....	.....	27	43	8	15	29	4	28	6	13
White spring																										
Baart 38	11907	3	11	0	86	76	90	88	74	4	1	2	91	4	4	72	0	2	10	0	96	94	91	0	10	1
Federation	4734	50	60	40	88	20	37	82	12	82	82	1	76	6	0	8	88	45	75	78	40	57	80	60	6	76
Federation 41	12230	10	9	0	90	91	83	90	74	5	0	0	75	2	2	50	0	7	13	1	86	89	70	1	1	0
Flomar	11707	16	20	24	48	35	30	32	26	1	12	60	0	20	0	5	0	40	28	35	31	42	44	45	1	14
Hope × Federation	11921	63	6	16	7	30	21	28	45	27	3	13	25	10	12	17	11	56	57	58	12	15	20	79	3	32
Hope × Federation	12424	4	1	6	5	13	3	5	18	1	2	2	8	3	2	1	2	3	9	9	5	1	3	1	2	2
Marflum	11918	11	1	9	13	25	14	29	17	1	1	41	1	39	0	1	1	15	21	14	22	20	6	27	69	75
Marquis-Flor. × Fed.	11919	40	3	4	49	71	62	26	33	0	7	85	1	79	1	3	2	68	71	52	19	57	21	50	64	48
Onas 41	12229	0	3	0	90	94	85	83	74	0	0	0	61	0	0	73	0	0	27	1	89	93	81	0	0	5
Orfed	11913	1	0	0	0	0	0	1	0	0	0	0	0	0	0	2	61	12	0	5	0	0	8	88	1	5
White Fed. 38	11906	2	4	0	85	88	88	82	67	1	0	0	67	1	0	61	1	3	17	0	90	86	76	0	0	0
White winter																										
Brevon	11912	2	4	4	4	19	4	13	3	2	5	74	1	89	3	2	0	17	13	5	5	3	10	5	72	59
Hymar	11605	0	0	0	54	64	56	90	68	0	0	0	50	2	0	29	0	0	0	0	70	84	61	0	0	0
Requa	11554	0	0	76	51	83	38	87	88	85	8	36	12	40	T	70	0	T	37	75	58	56	90	56	31	69
Rex	11689	1	1	0	28	22	51	71	69	0	0	0	23	1	0	96	0	0	1	0	66	42	50	0	0	0
Turk. × Florence	10080	0	0	6	0	14	0	0	T	0	T	38	0	28	0	0	0	.....	0	T	0	0	1	2	79	41
Washington Sel. 6	12241	0	0	0	0	0	0	0	0	0	0	7	0	2	0	0	17	0	0	0	0	0	0	21	5	T
T. durum																										
Mindum	5296	37	43	17	12	26	18	36	35	15	11	23	14	2	16	11	35	28	30	21	32	29	11	13	13	22
Carlton	12064	65	87	49	29	79	65	77	63	39	55	14	26	17	46	3	35	59	63	62	54	57	9	11	7	49
Stewart	12066	10	79	1	4	21	6	26	25	13	39	15	12	27	22	12	34	23	13	11	15	13	1	4	1	11
T. timopheevi	11802	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
T. timopheevi × *T. aestivum*																										
Sel. H143–1–1–8–110	12416	3	23	2	6	1	0	3	4	4	6	12	12	10	1	4	6	6	7	1	0	1	28	1	42	7
Sel. H143–1–1–13–8	12417	30	1	25	2	13	11	16	7	6	22	7	16	15	0	10	15	7	19	4	13	16	11	16	17	23
Sel. H143–1–1–3–13	12418	1	11	1	1	2	3	2	2	22	0	26	2	33	1	2	2	2	9	1	5	2	.....	2	28	2
Sel. H143–1–1–1–1	12419	13	11	2	2	0	2	0	10	42	6	38	4	34	1	2	3	3	6	2	5	8	12	1	35	12
Sel. H143–1–1–16–13	12420	2	8	9	0	4	1	3	0	16	12	35	2	36	2	5	9	6	0	1	34	11	41	1	15	2

common one in Mexico. The new races L–14 and L–15 have wider host ranges than L–2 and were found 7 and 4 times, respectively, in Mexico. It is also noteworthy that L–8, which, in the United States, was found only among the winter wheats of the western region, was collected once as far south as Puebla.

VARIETAL RESISTANCE

The varietal tests with different races of bunt reported in this paper were made to obtain information that would assist plant breeders in selecting parent material to use in the development of bunt-resistant varieties. The varieties tested include a number of commercially grown spring and winter wheats, some hybrid selections that in preliminary experiments had resistance to at least some of the known races, and a few varieties now being used as breeding material for rust resistance. The technique employed in preparing, inoculating, and planting the seed was the same as that previously described for experiments made in determining physiologic races. The spring wheats were tested at Aberdeen, Idaho, and the winter wheats at Pullman, Wash. All of the tests were not made the same year and for this reason the reactions for all varieties are not strictly comparable. The data presented, however, were obtained in years when environmental conditions favored high infection as evidenced by the fact that each year the winter wheat check variety, Hybrid 128 (C.I. 4512),[7] had on the average more than 75 per cent bunt, and the spring wheat check, Ulka (C.I. 11478), from 85 to 99 per cent for all races to which it is known to be susceptible. Furthermore, there was excellent differentiation between races on the standard differential varieties, which were included each year in the same plots for comparison. The smut percentages are based on total number of culms, averaging 300 per 5-foot row for the spring varieties and 250 per 6-foot row for the winter ones. Fractional percentages of 0.5 or more were recorded as 1 per cent, those below this fraction as a "trace" (T).

With a few exceptions, the results of the varietal tests with physiologic races are summarized in table 5. The data on the following 13 varieties and selections are omitted because these varieties were all either intermediate or susceptible in reaction to each of the races, and, therefore, are of no value as parent material in breeding for resistance to bunt. These varieties are Cadet (C.I. 12053), Frondoso (C.I. 12078), Frontiera (C.I. 12019), Kenya (C.I. 12332), McMurachy (C.I. 11876), Red Egyptian (C.I. 12345), Akron No. 7 (C.I. 11660), Tenmarq (C.I. 6936), Carlson's Fife (C.I. 11922), Elgin (C.I. 11755), Hindi (C.I. 8454), Jenkin (C.I. 5175), and No Name (C.I. 12301).

It is evident from the data presented in table 5 that there are a number of wheats that possess factors for resistance to all of the races of both species of the bunt fungi insofar as the races have been definitely delimited. There are five hard red spring wheats, four hard red winter wheats, and *Triticum*

[7] C.I. refers to accession number of the Division of Cereal Crops and Diseases.

timopheevi that are generally resistant. In the other groups, no generally resistant varieties have been found.

The five generally resistant hard red spring wheats are (1) Hope, (2) Komar × Hussar selection, (3) Regent × Pilot selection, (4) Reliance-1018 × Mercury selection, and (5) Renown. Except for Komar × Hussar selection, the resistance in these wheats, when sown in the spring, is apparently conditioned by the Hope factor. It is known, however, that certain spring wheat varieties are more resistant when planted in the spring than in the fall (3, 9, 11, 12). Hope is in this group and although it and the hybrids that have this variety as one of the parents were highly resistant when planted in the spring at Aberdeen, Idaho, it is quite possible they will be susceptible if planted in the fall. The high degree of resistance of the Komar × Hussar selection may possibly be due to transgressive segregation, as neither of the parents, which have been tested extensively in Uniform Bunt Nursery experiments with composite inoculum of some of the races, have had the resistance shown by the hybrid in these tests.

The four generally resistant hard red winter wheats are selections of (1) Oro × Turkey-Florence, (2) Rex × Oro, (3) Rex × Rio, and (4) Rio × Rex. It is also noteworthy that a selection of H–44 × Minturki (C.I. 12414) was resistant to all races except T–15 and T–16, to which it was intermediate in reaction. In general, however, it appears that both Hope and H–44 are less satisfactory as sources of bunt resistance for breeding winter wheats than they are for spring wheats. Thus in preliminary experiments at Pullman, Wash., in 1942, selections from 16 hybrid lines involving Hope with the winter wheats Cheyenne, Kanred, Mediterranean, Kawvale, Red Rock, and Fultz, and 8 lines of H–44 with Minturki were tested with a composite of races L–2, L–8, and T–8. One selection from the Hope hybrids and three selections involving H–44 were intermediate in reaction while the other 13 were completely susceptible.

Of the white spring wheats, a selection of Hope × Federation (C.I. 12423) showed some degree of resistance to all races and Orfed was highly resistant to all races except T–16 and L–8. Of the white winter wheats, Washington Selection 6, was intermediate in reaction to T–16 and L–8 and resistant to all the other races.

Unfortunately the new races from Mexico were not used in the tests. However, the resistance of Ridit and Oro to these races (Table 1) indicates to some extent how certain other varieties might react, and insures the availability of a resistance factor for developing new varieties resistant to these races should they become a menace to wheat production in the United States.

Triticum timopheevi (C.I. 11802) was the only wheat tested that was free from infection by all races. Shands (10) has succeeded in hybridizing *T. timopheevi* and a common wheat *T. aestivum* and five of his selections were included in these tests. The high degree of general resistance of the *T. timopheevi* parent, however, was not recovered in the hybrids. Each selection was resistant to some races, although the different selections were not always similarly resistant, intermediate, or susceptible to the different races.

Owing to the nature of the dwarf bunt fungus, pathogenicity tests comparable with those of the other races could not be made. There is evidence, however (5, 8, 14), that the so-called Hussar, Ridit, and Martin factors effectively control resistance to this bunt. Several hybrid strains deriving their resistance to dwarf bunt from one or more of these wheats were used in the present experiments, namely, Relief (Hussar × Turkey, Utah No. 26), Cache (Ridit × Utah Kanred), Hymar (Hybrid 128 × Martin) and Wasatch (Ridit × Relief). As would be expected from their parentage, these hybrids were resistant to a number of races but developed considerable bunt when inoculated with those races to which Hussar, Ridit, and Martin are susceptible. Oro × Turkey-Florence selection, on the other hand, is not only highly resistant to all of the races of ordinary bunt, but also has been resistant to dwarf bunt in several tests (5). However, the results have been somewhat conflicting inasmuch as this variety developed 22 per cent of dwarf bunt in one of the tests at Logan, Utah. Actually it probably has the same resistance to this bunt as Ridit and Cache, which is not adequate in areas of severe infestation. Theoretically, the variety combinations which offer the best possibility for producing resistance to all races, including dwarf bunt, are those that bring together the Oro and Martin types of resistance, which are carried by Rio and Rex, respectively. Data in table 5 on three hybrids of this type, namely, Rex × Oro, Rio × Rex, and Rex × Rio, show that they are highly resistant to all the races of ordinary bunt. Preliminary results[8] indicate that Rex × Oro and Rio × Rex are also highly resistant to dwarf bunt but no dwarf bunt tests have been made with Rex × Rio.

DISCUSSION AND CONCLUSIONS

Several factors probably operate to determine the distribution and relative prevalence of races of *Tilletia caries* and *T. foetida*. Among them, no doubt, are the direct effects of environment on the pathogen, on the host, and on the interrelation of the two as well as the distribution of wheat varieties. Regarding the influence of environment on the host, there is evidence that some varieties vary greatly in their reaction to certain races under different environmental conditions (7, 9). Obviously this factor might effect a build-up of inoculum of certain races in some localities. However, this probably is a minor factor, since the response of other varieties to the same races appeared to be unaffected by change in environment. Probably the distribution of specific wheat varieties is the most important factor governing the distribution and relative prevalence of the different races. Flor (2) found, in his survey of races of *Tilletia foetida* and *T. caries* in the Pacific Northwest, that the less virulent races of both species predominated on the old commercially grown varieties. While the more virulent races were found occasionally on the old varieties, they were more common in the districts where such varieties as Albit and Ridit were grown. From the results of the present survey, a similar condition appears to prevail in all wheat-growing

[8] Unpublished data from the Cooperative Uniform Dwarf Bunt Nurseries of 1944.

areas of the United States. Thus, for example, although L–8 was collected in California and in Puebla, Mexico, on varieties of wheat that are susceptible to all of the known races, it has become increasingly prevalent in certain areas of the Northwest where Oro and Yogo are widely grown. Similarly, race T–2 was found in such widely separated areas as Wyoming, North Dakota, and Georgia, but its importance coincides with the distribution of the durum wheats in the hard red spring wheat area. More recent changes in the varieties grown in certain other wheat-growing areas may afford opportunity for further study of this factor as it affects the distribution of races. For example, in the Southwest, Baart and White Federation, which are susceptible to the predominating races (L–1, L–2, L–3, and T–1) in that area, are being replaced by Baart 38 and White Federation 38. These new varieties should eliminate these races but, on the other hand, they may favor an increase in prevalence of the so-called Martin races to which they are susceptible. Likewise, it will be of interest to note whether the distribution of the new Turkey type wheats, Nebred and Comanche, will effect a build-up of race L–8 in the Nebraska and Kansas hard red winter wheat area.

It is evident from the study of the bunt reaction of different varieties (Table 5) that there is ample bunt-resistant breeding material. At the present time the Hope factor seems adequate to control the known races of bunt infecting spring wheats while combinations of factors from Oro with Martin or Ridit control the races that infect the winter wheats. For example, factors governing the so-called Martin reaction in the variety Rex when combined with those governing the Oro type reaction of the Rio parent gave a hybrid resistant to all races. Likewise, combining the Oro factors with those of Turkey-Florence, which supplied the so-called Ridit reaction, gave equally satisfactory results. Hybrids between Martin and Ridit were not available for the present studies. It should be possible, however, to derive from such crosses lines that would be resistant to all races since none of those infecting Martin infect Ridit, and vice versa.

The problem of breeding bunt-resistant varieties for certain sections of Utah, Idaho, Montana, and Washington is complicated by the occurrence of the dwarf bunt. The commercially grown varieties, Relief, Cache, and Wasatch, are resistant to dwarf bunt but are susceptible to certain races of both *Tilletia foetida* and *T. caries* that occur in those areas. There remains, therefore, the problem of combining the factors governing resistance to dwarf bunt with those governing resistance to races of both species of the ordinary bunt. Variety combinations that, theoretically, should produce this type of resistance would include as one parent, Rex, Hymar, Relief, or Wasatch, and as the other parent, Oro, Rio, Yogo, or Nebred.

SUMMARY

Seven new races of *Tilletia caries* and *T. foetida* are described, making the total number of known races 31, of which 16 are of the former species and 15 of the latter. Three of the new races of *T. foetida* were identified from collections made in Mexico.

Evidence corroborating results of previous surveys was obtained relative to the general distribution of the two species of the bunt fungi. *Tilletia foetida* is now the predominant species throughout the United States but *T. caries* is found over a considerable area. It is common in the durum section of the Upper Mississippi Valley and greatly increases in relative prevalence toward the intermountain area of the Northwest where it formerly was the predominant species.

The distribution of 16 races identified from 62 collections of *Tilletia caries* and 15 races from 307 collections of *T. foetida* are shown. Several of the races are widely distributed while others are more limited in their distribution. It seems evident that the distribution of specific wheat varieties is the most important factor governing the prevalence of specific races within a given area. Thus, for example, races T–11 and L–8 have assumed increasingly greater importance in the areas where Ridit and Oro, respectively, have replaced the old commercial varieties that had served as hosts of the more common races.

A number of varieties and selections were resistant to all of the races of ordinary bunt that were tested. It seems evident that thus far the Hope factor adequately controls these races in the spring wheats while lines derived by combining the so-called Oro and Martin factors, as in Rex × Oro and Rio × Rex, supply the necessary resistance for the winter types. Theoretically, selections from crosses between Martin and Ridit should likewise possess the desired resistance.

Dwarf bunt is a real menace to wheat production, particularly in certain sections of Utah, Idaho, and Montana. It is increasing in importance in the State of Washington and has been found in Wyoming, Colorado, and New York. The varieties, Relief, Cache, and Wasatch, although resistant to dwarf bunt are susceptible to a few races of the ordinary bunt fungi, hence there remains the problem of combining the factors that will supply resistance to both types.

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NEW PHYSIOLOGIC RACES OF USTILAGO HORDEI[1]

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INTRODUCTION

The importance of physiologic races of covered smut (*Ustilago hordei* (Pers.) Lagerh.) in the breeding and distribution of resistant varieties of barley in the United States is well recognized. In a previous paper (10) the writer reported the isolation of 8 clear-cut races among 200 covered smut collections from 26 States. Since the publication of that paper, 244 additional collections from barley-growing States have been studied. Five new races have been identified. The reactions of these races on the differential varieties and the distribution of races in the States from which collections were obtained are presented herewith.

PREVIOUS INVESTIGATIONS

The writer's earlier report (10) reviewed the previous studies up to 1937. Since then, Allison (1) in a one-year test of 28 collections on 11 varieties of barley found evidence of pathogenic differences in 27 of the lot but hesitated to consider all of them as distinct races. Semeniuk (9) studied 12 Canadian collections of *Ustilago hordei* and obtained evidence of 4 races, but in a later test the distinctions between 3 of the races had practically disappeared.

MATERIALS AND METHODS

The tests were conducted under field conditions at Ithaca, N. Y., 1939 to 1942, inclusive. The technique of seed inoculation, seeding, and other phases of experimentation followed that described in the report of the earlier studies at Ithaca (10). The pure-line differential varieties likewise were the same except that the black seeded variety Gatami (C.I. 575) was replaced by Himalaya (C.I. 1312). Smutted heads, especially the partly smutted ones, are difficult to detect in Gatami. A total of the 244 collections from 27 States were studied.

The seed used in each year of the test came from the previous season's crop produced under irrigation at Aberdeen, Idaho. This practice also was followed in the writer's previously reported tests (10) although a statement to this effect was not included. Judged by the results of Holton and Heald (5) with bunt in wheat and of Tervet (14, 15, 16) with loose and covered smuts in oats, the use of barley seed lots produced at one place in the same

[1] Cooperative investigations of the Division of Cereal Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture, and the New York (Cornell) Agricultural Experiment Station.

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season probably is important in physiologic race studies of the seedling infecting barley smuts *Ustilago hordei* and *U. nigra*. These investigators found that the various lots of a variety grown in different localities in the same year often show wide differences in susceptibility when artificially inoculated. Tervet (14, 15, 16) also obtained similar results with the various lots of varieties grown in the same locality in different years.

EXPERIMENTAL RESULTS

New Physiologic Races

Five new races (numbered 9 to 13) have been identified by their reactions on the 8 differential varieties during the 4 years 1939–1942. The

TABLE 1.—*Average percentages of smutted heads in 8 varieties of spring barley inoculated with 13 physiologic races of Ustilago hordei and grown at Ithaca, New York, 1934–42*

Race No.	Number of years tested	Average percentages of smutted heads in							
		Excelsior C.I. 1248	Hannchen C.I. 531	Himalaya C.I. 1312	Lion C.I. 923	Nepal C.I. 595	Odessa C.I. 934	Pannier C.I. 1330	Trebi C.I. 936
1	7	0.0	20.0	0.0	0.0	0.0	39.0	0.0	6.0
2	7	0.6	0.3	0.0	14.0	50.0	34.0	0.0	0.6
3	7	24.0	0.0	0.0	12.0	50.0	42.0	0.6	0.1
4	7	0.4	8.0	12.0	0.0	35.0	30.0	13.0	13.0
5	7	0.0	0.0	0.0	16.0	0.0	38.0	0.0	24.0
6	7	0.0	28.0	0.3	22.0	0.0	45.0	0.0	40.0
7	6	1.0	0.0	0.0	0.0	33.0	34.0	0.3	5.0
8	6	0.0	0.0	0.0	0.0	0.2	37.0	0.0	0.0
9	4	13.0	10.0	0.0	6.0	40.0	32.0	0.3	0.0
10	4	0.0	13.0	10.0	11.0	43.0	26.0	14.0	11.0
11	4	0.0	27.0	11.0	24.0	13.0	39.0	0.0	43.0
12	4	Trace	10.0	12.0	0.0	50.0	39.0	0.8	0.5
13	4	23.0	16.0	3.0	14.0	37.0	37.0	0.0	18.0

average percentages of infection obtained on the varieties with these races are in table 1. Included in the table are the averages of the previously isolated races 1 to 8 during the 1939–1942 period and the earlier years in which they were carried (10).

Each of the 5 new races (9, 10, 11, 12, and 13), as shown in table 1, had a distinctive pathogenic action.

Most of the collections of covered smut produced clear-cut reactions when first tested and were evidently composed of but a single race. Not infrequently, however, the reaction of the differential hosts indicated that a small admixture of one or more other races might be present. With the aid of screening hosts, 2 races were isolated from some collections and from an Arizona collection 3 races were obtained including the first isolation of race 13. In barley covered smut, at least thus far, there has been no evidence such as reported in oat smuts (8) and wheat bunt (2, 6) of spontaneous

origin of new races from previously stable races through mutation or segregation of factors for pathogenicity in heterozygous spores.

Minor differences in size and color of chlamydospores, smoothness of spore walls, compactness of the smutted heads and of the spore masses, degree of destruction of awns, and degree of exsertion of the smutted heads from the boot are not infrequently associated with differences in the pathogenicity of physiologic races of *Ustilago hordei*. The heads smutted by race 12 for example, are less compact and more spongy than those smutted by other races; they usually break out of the boot, as shown in figure 1; and the spore walls of the chlamydospores are slightly echinulate. Other investigators (4, 6) have previously reported similar variations in physiologic races of other cereal smuts. The magnitude of these differences is, in general, insufficient for definite identifications.

From the behavior of the 8 differential hosts, it is evident that even from this limited selection of varieties the control of covered smut through breeding for resistance should not be too difficult. The variety, Pannier (C.I. 1330), for example, was appreciably susceptible only to the infrequently collected covered smut races 4 and 10 and to date it has also proved immune from all 7 of the known physiologic races of the black or shallow-borne barley loose smut caused by *Ustilago nigra* (11, 12).

Finally, in connection with the data of table 1, it is interesting to recall that getting high percentages of covered smut in susceptible barleys under field conditions through artificial inoculation of the seed was formerly a difficult problem. The consistently satisfactory infections obtained through use of the spore-suspension method of seed inoculation (13) during the 7 years of these tests afford ample proof of the effectiveness of this method.

Stability of Races

In studies of differential spring wheat varieties that were inoculated with different physiologic races of the bunt fungi (*Tilletia levis* and *T. tritici*) and grown at different experiment stations, Rodenhiser and Holton (7) found that the susceptibility of a certain variety to a certain race in a particular year, for example, might be uniform at the different stations but the susceptibility of some other variety to this race might be relatively high at some stations and low at others. Similar results were obtained in the reactions at individual stations in the course of several years. Rodenhiser and Holton were inclined to the view that under the different environmental conditions the expression of genetic factors for protoplasmic resistance in certain varieties to certain races may be modified. During the course of the present study with barley covered smut at Ithaca, New York, a wide variety of climatic conditions was encountered but the relative susceptibilities of the differential varieties to each of the 13 races appear to have been adequately maintained. With race 6, for example, as shown in table 2, the varieties Excelsior, Himalaya, Nepal, and Pannier maintained their status of immunity or near immunity throughout the test. The per-

centages of smut in the susceptible Hannchen, Lion, Odessa, and Trebi varied widely under the different yearly conditions but each variety still

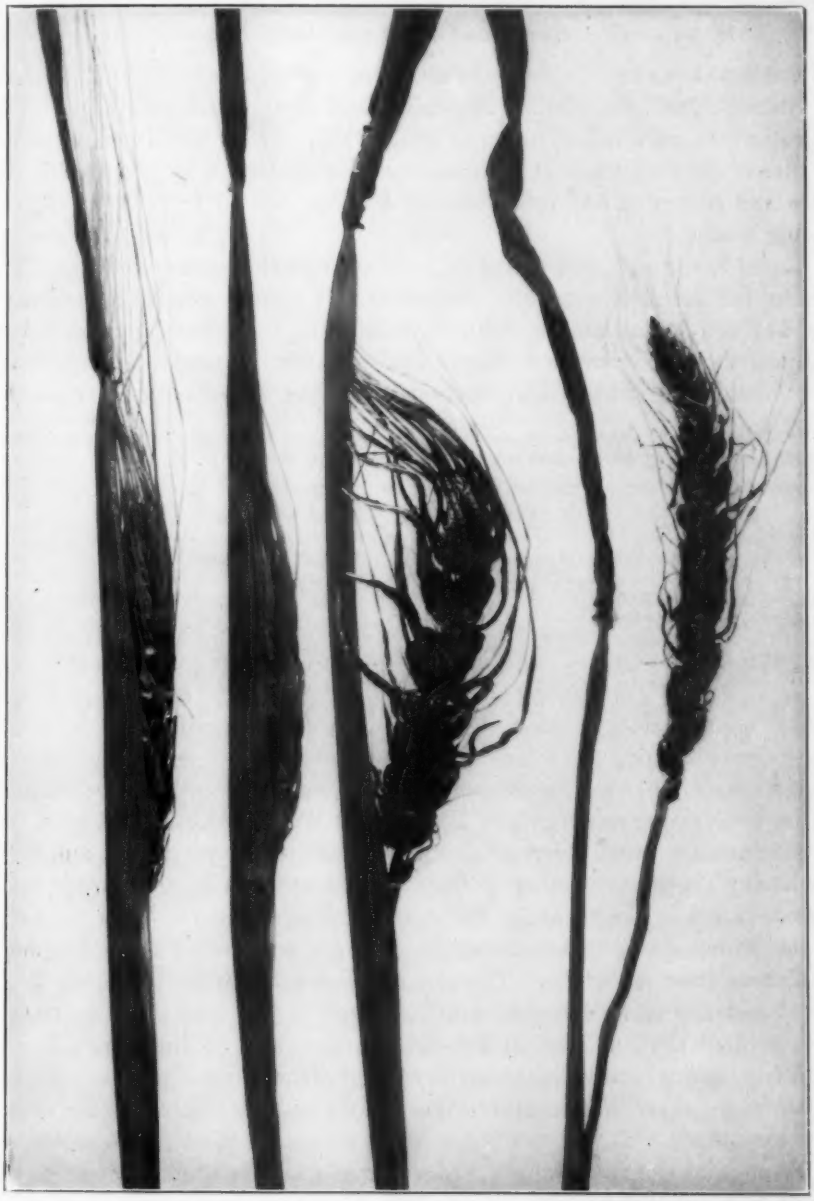

FIG. 1. Heads of Odessa (C.I. 934) barley smutted by covered smut; the two heads at left by race 7, the two at right by race 12, showing differences in morphologic expression.

maintained its relative degree of susceptibility. With few exceptions in the 7-year test, Hannchen was more susceptible to race 6 than Lion; Trebi was

more susceptible than Hannchen; and Odessa was more susceptible than Trebi. Similar general consistencies in reaction occurred with the other races.

Occurrence and Distribution of Races

Table 3 shows the frequency of occurrence of races and their distribution by States. The data include 244 collections of the present study and 200 collections of the similar, previous study (10). This gives a comprehensive picture of the frequency of occurrence and distribution by geographic divisions and States of 444 collections of *Ustilago hordei* from 33 barley-producing States.

Races 1, 5, and 6, comprised 86.5 per cent of the total collections. Race 6 is by far the most generally important. It occurred in 61.3 per cent of the 444 collections and is widely distributed. Race 5 appears largely confined to the far western States but it is very important in California and Washington where it comprised 63 of the 74 collections from these

TABLE 2.—*Percentages of smutted heads produced by physiologic race 6 in 8 differential varieties of barley over a period of 7 years at Ithaca, N. Y.*

Year of test	Percentages of smutted heads in							
	Excelsior	Hannchen	Himalaya	Lion	Nepal	Odessa	Pannier	Trebi
1934	0.0	30.0	.....	26.0	0.0	53.0	0.0	46.0
1935	0.0	17.0	.....	20.0	0.0	34.0	0.0	30.0
1936	0.0	32.0	.....	19.0	0.0	50.0	0.0	31.0
1939	0.0	42.0	0.0	28.0	0.0	51.0	0.0	60.0
1940	0.0	23.0	1.0	16.0	0.0	46.0	0.0	38.0
1941	0.0	21.0	0.0	15.0	0.0	31.0	0.0	31.0
1942	0.0	33.0	0.0	27.0	0.0	51.0	0.0	42.0

States. Race 1 has a wide distribution and was frequently collected in the winter-barley area as defined by Harlan and Wiebe (3). There seems to be some relation between the regions chiefly inhabited by races 1, 5, and 6, and the barley types grown there. Race 1 predominates in the winter barley region, race 5 in the region of Coast type barleys, and race 6 in the region of the Manchuria-Oderbrucker type. Race 8 was found more frequently in Kansas than elsewhere. The remaining 9 races were infrequently collected and together comprised approximately 9 per cent of all collections. It is evident that, at least at present, certain races of the barley covered smut fungus are highly important in certain areas while others are relatively unimportant there. This information should facilitate breeding for covered smut resistance.

Finally, as shown in table 3, from 1 to as many as 6 different races were found in individual States.

SUMMARY

Thirteen physiologic races of *Ustilago hordei* have been isolated, 5 in the present study of 244 collections and 8 in a similar previous study of 200 collections.

The combined results, involving 444 collections from 33 States, show that the widespread race 6 comprised 61.3 per cent of the total collections. The 3 most prevalent races, 1, 5, and 6, comprised 86.5 per cent of the total. In the winter barley area race 1 appears to be somewhat more prevalent than

TABLE 3.—*Frequency of occurrence and distribution by Grouped States of physiologic races of Ustilago hordei in 444 collections from 33 States, 1934–1942*

States	Number of collections of race													Total collections
	1	2	3	4	5	6	7	8	9	10	11	12	13	
North Atlantic														
Maine	.....	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....	1
New York	.....	.....	.....	.....	.....	10	.....	.....	.....	.....	.....	.....	.....	10
Pennsylvania	2	.....	.....	.....	.....	6	.....	.....	.....	.....	.....	.....	.....	8
North Central														
Illinois	.....	.....	.....	.....	1	4	3	1	.....	.....	.....	.....	.....	9
Iowa	2	.....	2	.....	.....	37	.....	.....	.....	.....	1	1	.....	43
Kansas	.....	.....	1	.....	1	7	.....	8	.....	.....	.....	.....	.....	17
Michigan	.....	.....	.....	.....	.....	5	.....	.....	.....	.....	.....	.....	.....	5
Minnesota	2	.....	.....	.....	.....	41	.....	.....	1	.....	4	.....	.....	48
Missouri	7	.....	1	.....	.....	1	.....	4	.....	.....	.....	.....	.....	13
Nebraska	.....	.....	.....	.....	.....	11	.....	.....	.....	.....	.....	.....	.....	11
North Dakota	1	.....	.....	1	.....	30	.....	.....	1	.....	.....	.....	.....	33
South Dakota	1	.....	.....	.....	.....	29	.....	1	4	1	3	.....	.....	39
Wisconsin	1	.....	.....	.....	1	36	.....	.....	.....	.....	.....	.....	.....	38
South Atlantic														
Georgia	3	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	3
Maryland	1	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	1
North Carolina	2	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....	3
South Carolina	1	.....	.....	.....	.....	.....	.....	.....	1	.....	.....	.....	.....	2
Virginia	10	.....	1	.....	.....	1	.....	3	.....	.....	.....	.....	.....	15
West Virginia	.....	.....	.....	.....	.....	1	.....	1	.....	.....	.....	.....	.....	2
South Central														
Louisiana	.....	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....	1
Mississippi	.....	.....	.....	.....	.....	.....	.....	.....	1	.....	.....	.....	.....	1
Oklahoma	4	.....	.....	.....	.....	5	.....	2	.....	.....	.....	.....	.....	11
Tennessee	1	.....	.....	.....	.....	2	.....	.....	.....	.....	.....	.....	.....	3
Texas	1	.....	.....	.....	.....	2	.....	.....	.....	.....	.....	.....	.....	3
Far Western														
Arizona	.....	.....	.....	1	2	2	.....	.....	.....	.....	.....	.....	1	6
California	1	.....	1	.....	42	1	.....	.....	.....	.....	.....	.....	.....	45
Colorado	.....	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....	1
Idaho	.....	2	1	.....	2	14	.....	.....	.....	1	.....	.....	.....	20
Montana	.....	.....	.....	2	.....	2	.....	.....	.....	.....	.....	.....	.....	4
Oregon	.....	1	.....	1	1	.....	.....	.....	.....	.....	.....	.....	.....	3
Utah	.....	.....	.....	.....	1	14	.....	.....	.....	.....	.....	.....	.....	15
Washington	.....	.....	1	1	21	6	.....	.....	.....	.....	.....	.....	.....	29
Wyoming	.....	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....	.....	1
Total collections of each race	40	3	8	6	72	272	3	20	8	2	8	1	1	444
Percentage of all collections	9.0	0.7	1.8	1.4	16.2	61.3	0.7	4.5	1.8	0.5	1.8	0.2	0.2	

race 6. In California and Washington, home of the Coast type barleys, race 5 was by far the most prevalent, comprising 85.1 per cent of the 74 collections from these States. Elsewhere throughout the country where, in general, the Manchuria-Oderbrucker barley types prevail, race 6 usually surpassed all others.

From 1 to 6 races were found in individual States.

During the 4 to 7 seasons that each of the 13 races was tested at Ithaca, N. Y., the 8 differential varieties of barley maintained their relative susceptibilities to each of the races although the climatic conditions within and between seasons often varied widely.

Not infrequently associated with different physiologic races of *Ustilago hordei* are minor differences in size and color of chlamydospores, smoothness of spore walls, compactness of the smutted heads and spore masses, degree of destruction of awns, and degree of exsertion of the smutted heads from the boot.

Among the differential varieties, Pannier (C.I. 1330) showed promise as foundation stock in breeding for smut resistance.

Consistently satisfactory infections throughout the 7 years of these tests through use of the spore suspension-method of seed inoculation afford proof of the effectiveness of this method.

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A NEW SPECIES OF COLLETOTRICHUM ON VETCH[1]

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INTRODUCTION

During the past several years, routine observations have been made on the diseases of vetches (*Vicia spp.*) among which was one that in certain characters resembled the diseases caused by *Kabatiella nigricans* (Atk. and Edg.) Karak., *Colletotrichum viciae* Dearn. and Overh., and *Ascochyta spp.* The diseases caused by *K. nigricans* and *Ascochyta spp.* often were present along with the anthracnose under discussion in this paper and it now appears that they were sometimes confused with it. It was finally decided that a disease complex existed that needed clarification and an investigation was initiated, the results of which form the basis of this paper.

DISTRIBUTION AND ECONOMIC IMPORTANCE

Little information regarding the distribution and severity of the anthracnose reported herein is available at present, other than the limited observations made by the writer near Experiment, Georgia, and a collection made by Dr. C. L. Lefebvre at Quincy, Florida. Dr. B. B. Higgins has observed an anthracnose on vetches at Experiment, Georgia, for a number of years but has made no study of it.

Considerable defoliation of smooth and hairy vetches is caused by the fungus during rainy seasons. Stems are blackened, and some stems are killed; but it is only in wet seasons when the crop is grown continuously on the same land that the disease becomes sufficiently abundant to damage commercial plantings.

SYMPTOMATOLOGY

On the leaves the lesions characterizing this disease are somewhat circular, but they may be elliptical or angular when delimited by veins, the margins of the leaflets, or an adjacent spot (Fig. 1). Although lesions vary in size, they are commonly from 1 to 2 mm. in diameter. There may be one or many lesions on a leaflet: sometimes the points of infection are so numerous that adjacent lesions coalesce and a large portion or even the entire leaflet may be involved (Fig. 2, A). In early stages the invaded tissues are light green, sometimes whitish or yellow-olive.[2] Even at this early stage the lesion has usually reached its maximum size. The color gradually fades until it becomes light brown, gray, or nearly white. The central area, which makes up half or more of the diameter of the spot, is surrounded by

[1] Cooperative investigations between the Division of Forage Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture, and the Georgia Agricultural Experiment Station. Paper number 138, Journal Series, Georgia Agricultural Experiment Station.

[2] Ridgway, Robert. Color standards and color nomenclature. 43 pp., 53 color plates. (Washington, 1912.)

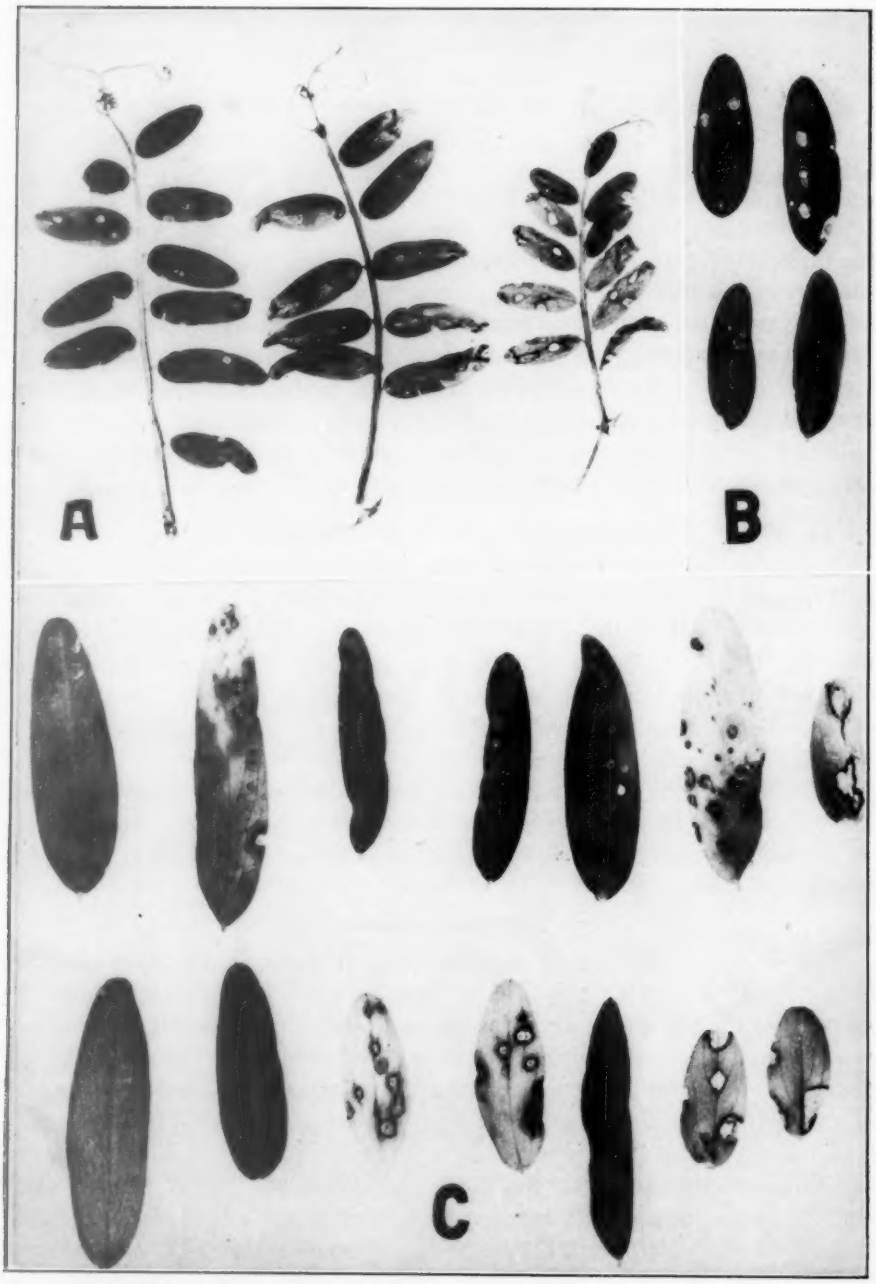

FIG. 1. Anthracnose leaf spot of vetch. A. Smooth vetch leaves showing shape and distribution of the spots on the leaflets. The red border surrounds the light center of the lesion or is adjacent to healthy tissue when the distal end or the margin of the leaflet is killed. ×1. B. Leaflets of purple vetch, upper and lower sides of the leaflets shown by the upper and lower leaflets, respectively. A second concentric ring having the color of the border is shown between the border and the center in some lesions. ×1. C. Leaflets of smooth vetch, the upper and lower sides of the leaflets illustrated in the upper and lower rows of leaflets, respectively. Two healthy leaflets at the left. The light leaflets had turned yellow. Dark conidial masses are in the centers of some lesions. ×1¾.

a narrow border that may be Brussel's brown, raw umber, bay, or warm sepia, depending on the variety of the host attacked and other factors. Even when large areas of the leaflet are involved and the outline of the spot is lost the discolored border is present, and sometimes veins are discolored when the entire leaflet is involved. Severely affected leaflets eventually turn yellow and fall off. The colored border is not evident in the very young lesions but is soon formed and gradually develops into a well-defined narrow band adjacent to the healthy tissue. Rarely it may be more diffuse and cover a large proportion of the lesion. Sometimes there is a secondary, usually lighter, concentric ring similar to the border but nearer the center of the spot (Fig. 1, B). Under moist conditions the central area contains what appears to the unaided eye to be a large globular mass of conidia (Fig. 1, C). Examination with the hand lens or low power of the microscope shows that this mass, which usually is from $\frac{1}{4}$ to 1 mm. in diameter, consists of many small acervuli, that are 25 to 100 μ in diameter, more commonly 30 to 50 μ. Acervuli may be scattered irregularly over the lighter parts of the lesion or may be so close together that the masses of conidia coalesce. They are most common on the lower leaf surfaces, especially those of the lower leaves which may be almost completely covered by acervuli during a prolonged wet period. Conidial masses at first are nearly white or gray but soon darken to an ivory yellow, and later to a warm buff or ochraceous buff. In older spots this central area may appear nearly black because of numerous setae and black stromatic cells. Leaves killed by the fungus may have many black stromatic masses on one or both sides of the leaflets.

On young stems and petioles the lesions, at first slightly sunken, are linear, light-green areas, from 1 to a few mm. long by less than 1 mm. wide. The lesions darken with age, enlarge somewhat, seldom become more than $\frac{1}{2}$ cm. long, and often coalesce with adjacent spots until large areas of the stem may be involved (Fig. 2, B and C). The centers of the stem lesions are light and the margins are much the same color as the borders of the leaf spots. When large areas of the stem are involved the color usually approaches a warm sepia. Acervuli are borne in the centers of the younger lesions. Young stems, petioles, and tendrils may be killed. Old stems are discolored and defoliated, but whether or not they are actually killed has not been definitely established. Young stems are seldom killed by a single lesion, but if two or more lesions occur near one another, as on opposite sides of the stem, a sufficiently large proportion of the tissue may be involved to girdle the stem. Small stromatic bodies may be present on the stems as well as on the leaflets. Setae may be present in varying number or they may be wanting.

The fungus has not yet been obtained from the pods, but it seems probable that pods are attacked.

PATHOGENICITY OF THE FUNGUS ISOLATED FROM LESIONS

Isolations from the spots on the leaves and from stems have shown the constant association of an anthracnose fungus with the disease. The fungus

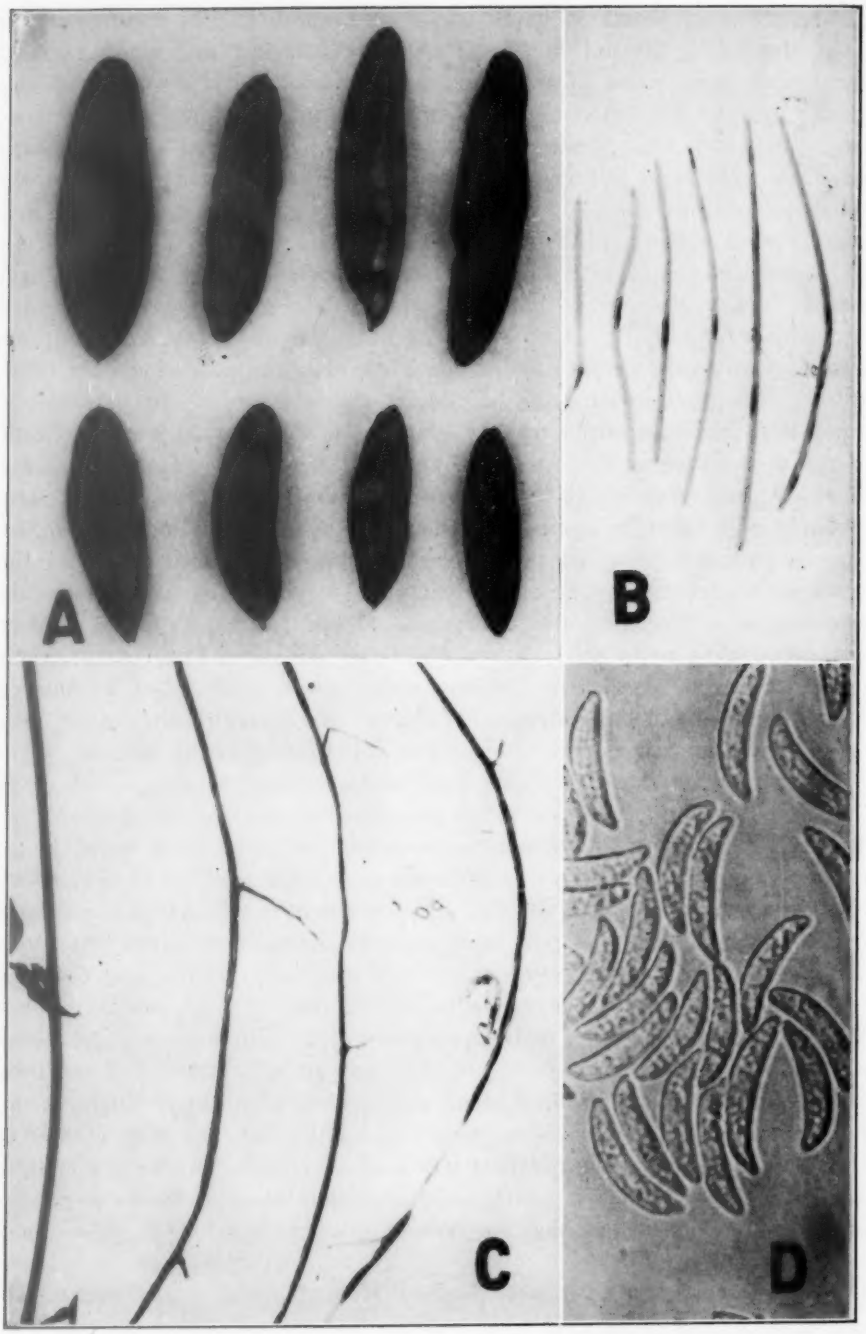

FIG. 2. A. Upper and lower sides of vetch leaflets showing early stage of infection under extremely favorable conditions. The light green lesions, with little or no colored border yet visible, often are confluent and may involve a large part of, or the entire, leaflet. ×2. B. Stems of smooth vetch seedlings with early stage of infection resulting from inoculation. ×2. C. Stems of older plants. Stem at the left is healthy, others have dark lesions resulting from inoculation. ×1⅞. D. Conidia of *Colletotrichum villosum*. ×1000.

has been grown in pure culture, single spore lines obtained, and inoculations made. The fungus obtained in culture was pathogenic on several species of vetch.

On October 4, 1943, young smooth vetch plants growing in steamed soil in pots in the greenhouse were atomized with a sterile tap water suspension of conidia from a pure culture. Control plants were atomized with sterile tap water. The plants were kept in an inoculation chamber at high humidity for 48 hours and then returned to the greenhouse bench. Six days later water-soaked spots were on some of the leaflets and soon became light green. Two days later the characteristic brown borders appeared about the spots, and typical conidial masses developed later. Some of the stems were heavily infected, while others had only a few lesions (Fig. 2, B and C). In one pot there were 61 seedlings, 24 of which had stem lesions. One month after the inoculations 4 seedlings had died from stem girdle and several others were nearly dead; whereas some showed little damage. When petioles were infected the leaflets usually died. The larger stems were seldom girdled by a single lesion, but two or more lesions on opposite sides frequently severed these seedling stems. Often there were many lesions on a stem. Isolated lesions were linear and not over 5 mm. long by about 1 mm. wide. When several lesions had coalesced, areas 10 cm. or more were sometimes involved. The lesions varied considerably in color, the borders being chocolate to almost black, and the centers light ochraceous salmon to nearly black. One to many black, raised fruit bodies, with diameters of 1–0.1 mm. or less, were scattered over the surface of the lesions.

Another inoculation experiment was started on October 18, 1943. The plants were over a month old, and were approximately the size of plants in the field when infection first becomes apparent in the spring. A heavy conidial suspension was made from two 10-day-old cultures of the fungus growing on corn-meal agar in Petri plates. Three potted plants were inoculated and one potted plant was maintained as a control. After being atomized with the conidial suspension, the plants were held for 40 hr. in a moist chamber, in which the initial temperature was 35° C. but lower at night. After 6 days the infection was evident as pale green, more or less circular, spots on the leaflets. Many leaflets were infected and a considerable number had fallen 3 days later, or 9 days after the inoculations. Many of the lesions had developed the characteristic brown borders at the junction of the diseased and healthy tissue. Some lesions had conidial masses in the center and these contained mature conidia. In many instances the healthy leaf tissue beyond the spot had turned yellow and the leaflets had fallen. Thus, considerable defoliation can occur and a new crop of mature conidia be produced within 8 to 10 days after inoculation. On the ninth day the stem lesions were linear, from one to a few mm. long, and less than 1 mm. wide. The lesions had one or more pustules bearing mature conidia. The fungus was readily recovered from the diseased tissue, grown in pure culture, and used in further inoculation tests.

POSSIBLE SOURCES OF INOCULUM

The earliest stage of the disease observed under natural conditions is the leaf-spot stage seen in early spring. Its presence in destructive amounts varies from year to year depending largely on the amount of rainfall and the amount of inoculum present. The plants are usually fairly well developed before much damage is done, but may be severely defoliated about blossoming time. The source of the early infections has not been definitely determined; but two experiments were conducted to determine if the fungus could be transmitted by seed or soil under experimental conditions.

Pots of soil were steamed and then each was planted with 25 seeds of smooth vetch. Two pots were planted with seed that were first immersed in a conidial suspension. A heavy suspension of conidia was applied to the soil in 2 other pots after the seed had been planted. Three pots were held as controls. The seeds were planted February 3, and the seedlings examined March 3, 1944. When removed from the soil, washed, and examined, 47 plants were in the 2 pots in which the seed were inoculated and all had brown to black discolorations of the stem just below the soil surface (Fig. 3, A). Distinct lesions were present, some of which were very minute, whereas others had coalesced and formed darkened areas of considerable size. No girdling had resulted. The 2 pots in which the soil had been infested had 22 plants and all had darkened stems as did those for which the seed had been inoculated. Only 2 of the 69 control plants had a somewhat similar blackening. Isolations were made from several of the blackened areas. Since lactic acid could not be used to inhibit bacterial growth, the fungus was rather difficult to isolate; and it grew so slowly that when secondary fungi or bacteria were present they invariably outgrew it. Since the infected parts were underground tissue, secondary organisms, largely Penicillia, Fusaria, and bacteria, were present in many of the platings. However, after the cultures were several days old, each plate was examined microscopically and both setae and conidia of the anthracnose fungus were found. Since inoculated seed had been planted in steamed soil and clean seed had been planted in soil infested with conidia of the fungus, and since the control plants had no infection, there seems to be little doubt that the anthracnose fungus, when present on the seed or in the soil, can attack the stems of the young seedlings. Conidia formed on these initial infections can supply inoculum for the continued spread of the fungus to other parts of the plants.

Many plantings of vetches have been observed and it seems likely that the fungus may live over in the soil or on plant débris, since the disease may be absent or present in very small amount when vetches are grown on land that has not previously grown this crop, yet it may be very severe when vetches are grown on land that has grown vetch for several consecutive years. It also is possible that the seeds become contaminated with conidia when diseased plants are threshed or that pieces of stem, leaf tissue, or thick-walled stromatic cells present on leaf blades, petioles, or tendrils might

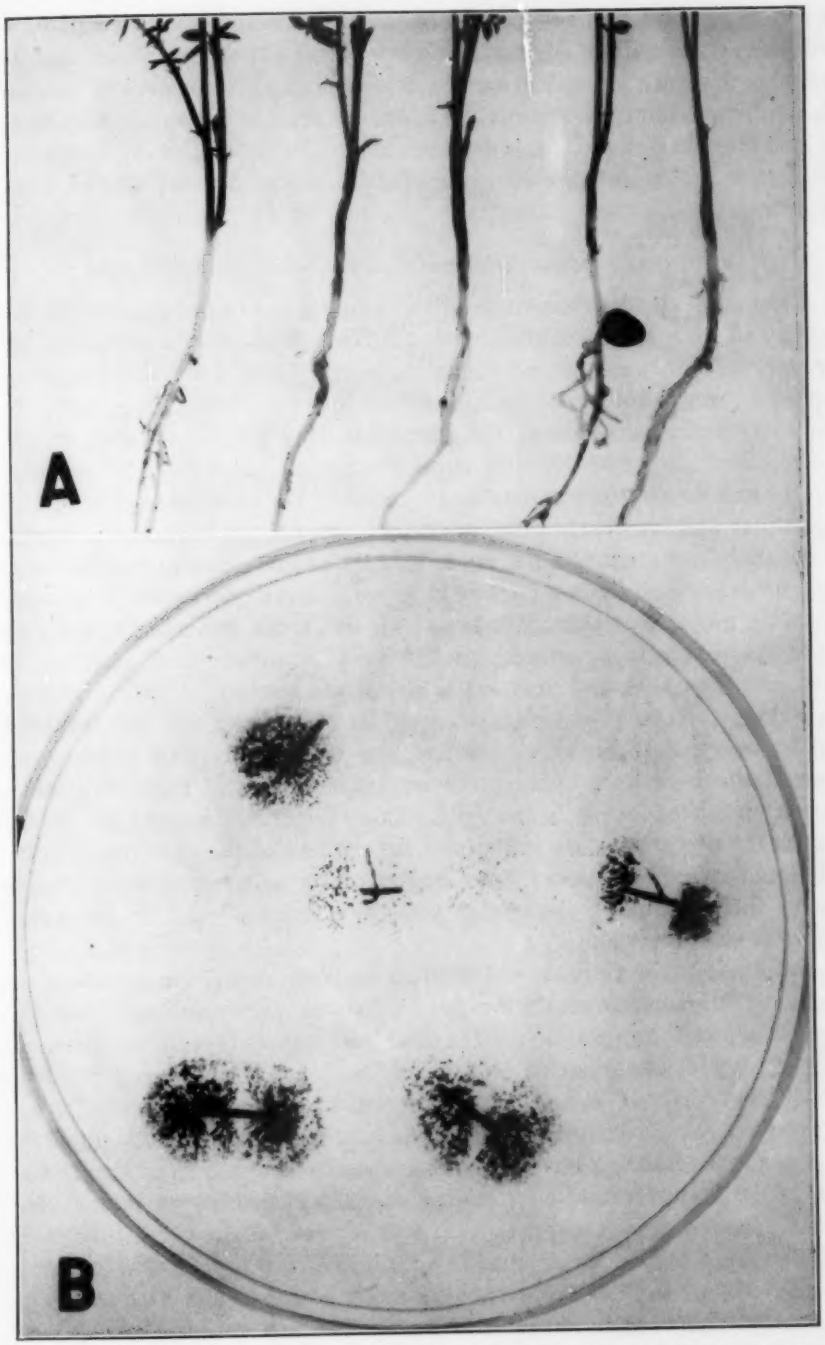

FIG. 3. A. Stems of young smooth vetch plants grown in pots in the greenhouse. The plant at the left is healthy, the next 2 plants to the right have dark lesions, on the stems just below the soil surface line, resulting from infection in artificially infested soil. The 2 plants at the extreme right have similar lesions resulting from seed inoculation. $\times 1\frac{1}{10}$. B. The fungus growing from surface-disinfected, diseased stems on corn-meal agar. $\times 1\frac{1}{12}$.

be mixed with the seed and carried to the field. Volunteer plants of smooth and hairy vetch often are widespread in vetch-growing sections and it is probable that such plants can harbor the fungus and be a source of inoculum. No perfect stage of the fungus is known. Since one crop of vetch matures in late May or June and the next is planted in September or October, no prolonged period for the survival of the fungus outside of the host is necessary.

CULTURAL CHARACTERS AND MORPHOLOGY OF THE FUNGUS

Some difficulty was experienced in isolating the causal fungus from the tissue and even from conidial masses. The fungus would not grow on a strongly acidified medium, as, for example, when 1 or 2 drops of 50 per cent lactic acid were added to 15 ml. of medium to inhibit bacterial growth. Several other anthracnose fungi, from legumes, grew readily on such an acidified medium. Some of the early studies were attempted during the summer months and no growth was obtained because of too high temperature. Once the acidity and temperature factors were corrected the fungus could readily be isolated from stem (Fig. 3, B) or leaf tissue and grown on different culture media. The fungus grew well and sporulated on corn-meal and oatmeal agars. Two per cent potato-dextrose agar and fresh, green-string-bean pods proved to be excellent media for production of conidia. Canned beans were much less favorable, and nutrient agar was unsuitable. At best the fungus grew slowly. It produced a white mycelium which later turned dark brown, and became thick-walled and much septate (Fig. 4, M). On old corn-meal agar slants there were many small irregular masses of dark, thick-walled cells often buried deeply in the agar. These sometimes were so abundant as to give the medium a dark color. On the surface of the agar many gray to pale-salmon conidial masses were formed, with or without setae scattered among them. One or more setae sometimes were present in or near the center of each acervulus.

Germination of conidia was studied in some detail on corn-meal-agar plates. Conidia from a 10-day-old culture on corn-meal agar were suspended in sterile tap water, then poured over the surface of the agar. The earliest sign of germination was noted in 4 hours at room temperature (25–30° C.). About 50 per cent of the conidia had germinated in 24 hours and germ tubes were from 1 to 5 times as long as the conidia. A single germ tube was formed at or near one end, more rarely at the side, of the conidium (Fig. 4). The germinating conidium usually became septate very early. In some cases a brown appressorium was formed on the germ tube a short distance from the conidium, and this later sent out a tube that penetrated the agar (Fig. 1, D). On corn-meal agar, however, the germ tube more commonly continued to elongate, and after 24 hours conidiophores began to develop. A small swelling first appeared on one side of the germ tube a short distance beyond the conidium (Fig. 4, E), continued to enlarge, and became a conidiophore varying in length from 1 to 5 times, but more often

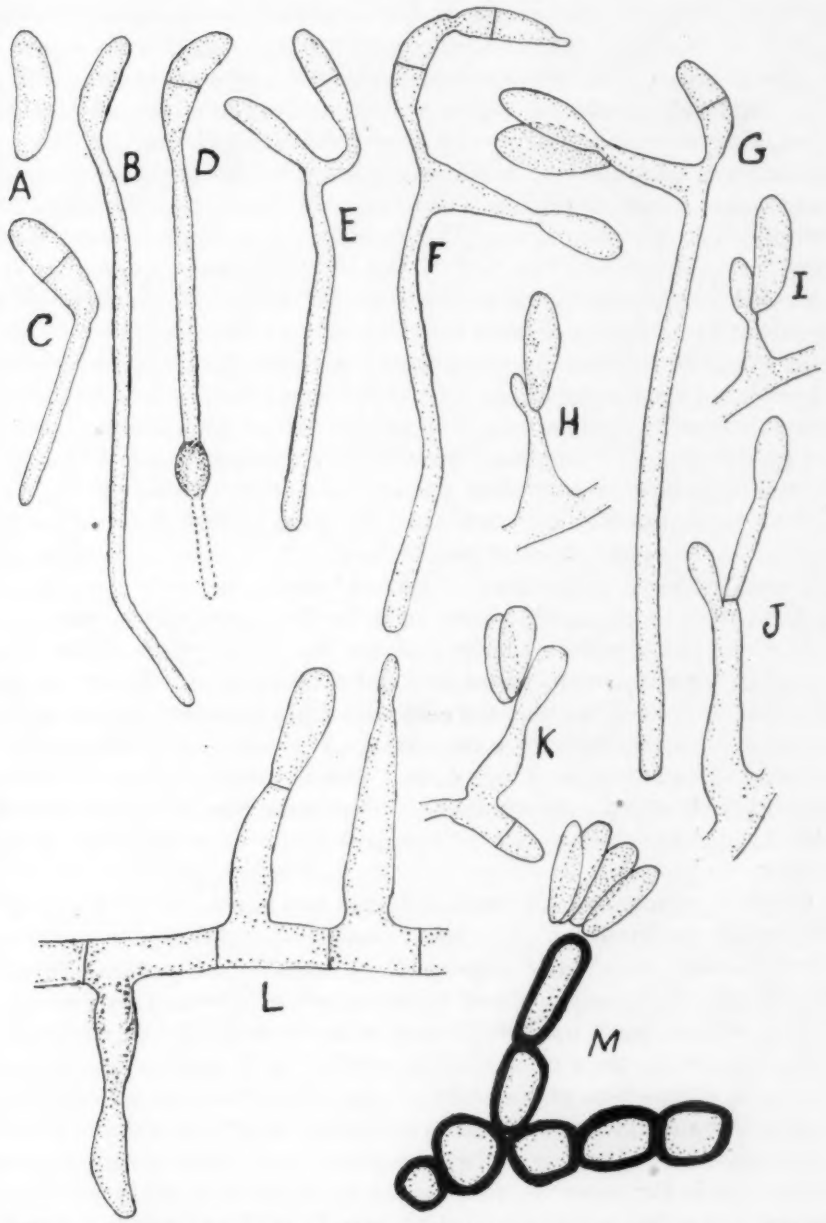

FIG. 4. Different stages of conidial germination on corn-meal agar. A. Not germinating. B, C, and D. Early stages of germination. D. An appressorium a short distance from the conidium. E. Conidiophore forming on the germ tube near the conidium. F. Slightly more advanced stage than in E. Conidium forming on the young conidiophore and a secondary conidium, also starting to germinate, formed directly from the other cell of the conidium. G. Slightly later stage than in F. The first conidium formed has been displaced by a second one. H, I, and J. The second conidium formed by a conidiophore pushes aside the first conidium which eventually falls off. K. Short conidiophore formed directly from one cell of the conidium. L. The multicellular germ tube, with each cell sending out a conidiophore which bears numerous conidia. M. Thick and dark walls of the old mycelium that forms black stromatic layers. Even the walls of the conidiophore are thick and dark. All drawings approximately × 1000.

2 to 3 times, as long as the conidium. The conidiophore usually was slightly wider at the base than the germ tube and often was slightly constricted at the point of attachment. It tapered to approximately the size of the conidium at the distal end. A single conidium formed at the apex of the conidiophore and as this matured a second conidium formed (Fig. 4, H and I). The first conidium was finally shoved aside and dropped off as its place was taken by the second conidium (Fig. 4, G and K). This process continued so that eventually a considerable number of conidia accumulated on the agar at the end of the conidiophore (Fig. 4, G, K and M), very much as described and illustrated for cotton anthracnose by Atkinson.[3] The germ tube continued to elongate and sometimes reached a length of 500 to 1,000 μ in 72 hours, and it finally branched to form a small mycelial mat in 90 hours. A conidiophore was produced from most of the cells thus formed, and each conidiophore formed numerous conidia. In this manner large quantities of conidia were produced as the result of the growth of a single conidium. As many as 50 conidiophores were counted on a single germ tube 72 hours old. After the first germ tube was well developed the other cell of the now septate conidium germinated. A short germ tube usually was formed and served as a conidiophore. Sometimes a typical conidiophore or a secondary conidium with no perceptible germ tube formed directly from either cell of the germinating conidium (Fig. 4, F and K).

Young mycelium was hyaline and 2–4 μ in diameter. Later the mycelium became closely septate, the cells were more rounded, sometimes 10 μ or more in diameter, the walls became thick and brown, and thus a stromatic mass of cells was formed (Fig. 4, M). Even the conidiophores became dark and thick-walled. Setae might be interspersed among the thick-walled cells. One of the thick-walled cells sometimes served as the basal cell of the seta.

Conidial germination was studied first in tap water. Short germ tubes were usually produced by only a small proportion of the conidia, and then growth ceased. Apparently some condition suitable for continued growth was missing. Since conidia placed in the same tap water and then atomized on the host gave good infection, it was thought that juice or particles of the host might stimulate growth of the conidia in tap water. This was the case, since when pieces of tissue or juice of several species of vetch were mixed with a suspension of conidia in sterile tap water considerable increase in germination and in vigor of the germ tubes was apparent. In some cases germination in the water controls was about 10 per cent; when vetch juice or tissue was added germination varied from 75 to 95 per cent.

EFFECT OF TEMPERATURE ON THE FUNGUS

A knowledge of the temperature relations of *Colletotrichum villosum* is essential if it is to be cultured successfully. The fungus failed to grow in the laboratory during the summer because of too high temperatures, and

[3] Atkinson, G. F. Anthracnose of cotton. Jour. Mycol. 6: 173–178. 1890–91.

cultures were lost unless held in a refrigerator. The following summary of the temperature studies will serve as an aid in culturing the organism.

The fungus was grown on 2 per cent potato-dextrose agar in Petri dishes held at different temperatures and the results were recorded at the end of 2 weeks. A few conidia germinated at 3.5° C., and at 13.3° C. germination was good but no macroscopically measurable colony had been formed. The upper limit for growth was near 32 and the optimum lay between 24 and 26° C. Growth was fair at 21.5 and 29° C.

DESCRIPTION OF THE CAUSAL FUNGUS

This anthracnose disease has certain characters in common with the disease caused by *Kabatiella nigricans*. The symptoms on the stems, at least to one not familiar with both diseases, can readily be confused. The conidia of the anthracnose fungus and those of *K. nigricans* are very similar both in size and shape. The leaf symptoms, however, are different. Lesions of *K. nigricans* are illustrated and described by Wolf[4] as "circular with a tendency toward being most numerous along the principal veins or . . . as elongated, dark streaks." A study of conidia production and germination shows clearly that the anthracnose fungus differs from *K. nigricans* in that in the former the conidia are borne singly on conidiophores, about the width of the conidium at the point of attachment instead of on the large conidiophore characteristic of *Kabatiella*. Likewise there is no budding of the conidia on germination as there is with *K. nigricans*.

The literature dealing with closely related diseases of vetch is reviewed by Wolf.[4] He mentions *Gloeosporium davisii* Ell. and Ev. and *G. everhartii* Sacc. et Syd. These possess smaller and differently shaped conidia than does the fungus studied by the writer. The same is true of *G. viciae* Fautr. et Roum. *G. tricolor* Lind has much longer conidia than does the fungus of this paper. The literature on the anthracnoses of other legumes has been surveyed and so far as could be ascertained all the causal fungi are different from the vetch anthracnose fungus considered here.

Perhaps the fungus most similar to the one studied by the writer is *Colletotrichum viciae* Dearn. and Overh. Dr. Overholts kindly sent the writer a specimen of his material and a study of this seems to leave little doubt that the two fungi are different. For example, *C. viciae* does not form definite circular lesions with colored borders on the leaflets but a part or all of the leaflet is affected, and acervuli are scattered promiscuously over the affected tissue just as described by Dearness.[5] The acervuli are more numerous on the upper side of the leaflets affected by *C. viciae*, which is just the reverse from that in the Georgia material. The conidia of the writer's species average about the same as the maximum width of *C. viciae*. The setae of *C. viciae* are shorter and narrower and have only 1 septum instead of 1 to 4. It seems to the writer that these differences in symptoms

[4] Wolf, F. A. A little-known vetch disease. Jour. Elisha Mitchell Sci. Soc. **36**: 72–85. 1920.

[5] Dearness, J. New and noteworthy fungi—V. Mycologia **20**: 235–246. 1928.

produced in the host together with the location of the acervuli most commonly on the upper side of the leaflets, the differences in conidial width, and in size and septation of the setae, are sufficient to justify the conclusion that *C. viciae* is a different species from the one studied by the writer.

Since the fungus attacking vetch in Georgia appears to be different from any described in the literature, it is considered a new species and the name *Colletotrichum villosum* is proposed.

TECHNICAL DESCRIPTION

Colletotrichum **villosum** n. sp.

Acervuli in foliis amphigeni, saepius hypophylli, 25–100 μ in diam.; massae conidicae griseolae usque ochraceo-bubalinae; setae carentes vel abundantes, hyalinae usque rubro-brunneae, 45–150 × 6.3–9.5 μ ad basin bulbosam, attenuatae, 1–4-septatae; conidia hyalina, subcurvata, unicellularia, 15–24 × 3.5–4.5 μ.[6]

Colletotrichum **villosum** sp. Lesions typically more or less circular to linear, light brown to gray with Brussel's brown to warm sepia border, becoming dark brown to almost black on stems, amphigenous, 1–2 mm. in diameter. Acervuli amphigenous, more commonly hypophyllous on leaflets, 25 to 100 μ in diameter, more frequently 30 to 50 μ, or larger and irregular when confluent. Conidial masses gray to warm buff to ochraceous buff. Setae wanting or abundant, variable from hyaline to purple-gray to red-brown, approaching Brussel's brown of Ridgway, becoming lighter at the tips and often at the base, average 85 μ (range 45 to 150 μ) long by 7.5 μ (range 6.3 to 9.5 μ) wide across the bulbous base, tapering to a rounded point, 1 to 4 septate. Conidia hyaline, slightly curved, bluntly tapered, unicellular, average 18.9 by 4 μ (range 15–24 × 3.5–4.5 μ) 18 to 20 μ most common length.

Habitat: Parasitic on leaves and stems of *Vicia villosa* Roth., *V. sativa* L., *V. atropurpurea* Desf., *V. grandiflora* Scop., *V. monanthos* (L.) Desf., *V. dasycarpa* Ten., *V. angustifolia* L., and *V. pannonica* Crantz in Georgia and Florida, U. S. A.

Material has been deposited in the herbaria of Mycological Collections, Bureau of Plant Industry, U. S. Department of Agriculture, Beltsville, Md., Department of Plant Pathology, Cornell University, Ithaca, N. Y., and Department of Botany, Georgia Agricultural Experiment Station, Experiment, Ga. No. 71420 (Myc. Coll. Bur. Pl. Ind.) collected by J. L. Weimer, Experiment, Ga., on *Vicia villosa* Roth, May 6, 1943, is designated as type.

CONTROL

Although little experimental work has been done on the control of this disease, several methods may be possible. The use of clean, disease-free seed, preferably obtained from areas where the disease does not exist, or where environmental conditions are not suitable for its rapid development, should reduce the possibility of distributing the fungus, or at least delay its accumulation in the soil of any field not already heavily infested. Evidence available at present suggests that the disease seldom becomes destructive if the crop is not grown on the same land continuously. This means that rotation properly managed may be a fairly satisfactory control measure. The destruction of volunteer vetch in the vicinity of vetch fields will eliminate another source of inoculum.

One means of controlling such field-crop diseases is the use of resistant varieties or closely related species that might be suitable as crops. In order

[6] Latin diagnosis supplied by Edith K. Cash, Division of Mycology and Disease Survey, Bureau Plant Industry.

to determine if any of the common vetch varieties or species were resistant, several lots were grown in pots in the greenhouse and inoculated by atomizing with a conidial suspension. The ratings of the different lots tested in two experiments were based on a 0 to 10 scale (Table 1). The lots of *Vicia sativa* were fairly resistant for the most part. They were not immune, since most of them had a large number of lesions, but these remained small

TABLE 2.—*The relative resistance of some vetch varieties and species*[a] *to anthracnose. Rated*[b] *on a scale of 0 to 10*

Variety or species name and F.C. or other number	Rating in Experiment 1	Rating in Experiment 2
Vicia sativa, Selection 5	2	3
7	1	2
103	5	.....
F.C. 16462	1	2
Vicia sativa alba, F.C. 02830	2	3
F.C. 18808	2	3
F.C. 81566	2	1
F.C. 18052	2	.....
F.C. 18056–1	2	.....
F.C. 18814	.....	4
F.C. 18818	1	.....
F.C. 22785	2	.....
F.C. 22790	2	.....
F.C. 29933	2	2
F.C. 30171	2	.....
F.C. 31361	3	.....
F.C. 31542	1	2
F.C. 31543	2	.....
F.C. 34947	2	4
F.C. 11725	2	.....
F.C. 119988	3	.....
F.C. 119994	1	4
F.C. 31546	1	3
F.C. 31545	1	1
V. atropurpurea Desf.	9	10
V. grandiflora Scop.	1	1
V. villosa Roth.	9	10
V. monanthos (L.) Desf., F.C. 18139	4	3
V. monanthos, strain not numbered	3	3
V. dasycarpa Ten., F.C. 22471	10	9
V. angustifolia L., F.C. 04315	2	4
V. pannonica Crantz	1	1

[a] The writer is indebted to Mr. Roland McKee, Division of Forage Crops and Diseases, Bureau of Plant Industry Station, Beltsville, Maryland, for all of the seed used.
[b] Rating: 0 = no disease; 5 = moderate disease; 10 = severe disease.

and superficial, and in many cases were confined largely to the stems. The only lots severely injured in these experiments were those rating 8 to 10, namely *V. atropurpurea*, *V. villosa*, and *V. dasycarpa*. Just what would have happened to the other lots had they been subjected to a long rainy spell, as often happens in the field, is not known. However, in hotbeds where these lots were outdoors for 5 months without artificial inoculation but growing in field soil in which vetch had been grown for several years, only *V. villosa* and *V. atropurpurea* were severely damaged by anthracnose.

Also, the lots of *V. sativa* have never been observed severely damaged in the field, even when *V. villosa* nearby was largely defoliated. It may be concluded, therefore, that it is possible to control vetch anthracnose by growing one of the lots of *V. sativa* rating 3 or less, or any of the other species listed in table 2, except the 3 rating 8 or above. At the conclusion of these experiments most plants in these 3 high-rating lots were dead, the stems as well as the leaflets having been killed. In a few cases where the stems were killed, new secondary stems were formed near the surface of the soil. Presumably in the field these shoots would have been infected and possibly killed if environmental conditions were suitable for the spread of the fungus.

SUMMARY

An anthracnose leaf spot of vetches is caused by *Colletotrichum villosum* n. sp. On the leaves the fungus produces small circular spots at first light green, later becoming light brown or gray with a brown to red border. The stem lesions are linear and usually dark to black. Severe defoliation and death of young stems may occur during wet weather. The fungus grows slowly on nonacid culture media and fruits abundantly on sterilized, fresh string-bean pods, and on potato-dextrose, corn-meal, and oat-meal agars. The optimum temperature for growth lies between 24° and 26° C., some growth takes place at 3.5° C., and the maximum for survival is near 32° C. *Vicia atropurpurea, V. villosum,* and *V. dasycarpa* are the most susceptible of the species tested, whereas most strains of *V. sativa, V. grandiflora, V. monanthos, V. angustifolia,* and *V. pannonica* are fairly resistant. The use of disease-free seed, rotation, and the use of resistant varieties are suggested as control measures.

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A LINE-PATTERN VIROSIS OF SHIRO PLUM[1]

R. S. WILLISON

(Accepted for publication July 14, 1945)

Early in the summer of 1938, symptoms suggestive of virus origin were observed on a number of trees of the Japanese plum variety, Shiro, in a 20-year-old block near Port Dalhousie, Ontario (3). The older leaves were marked with a creamy white vein-banding (Fig. 1, E), but the younger leaves were apparently normal. Symptoms persisted on the older leaves throughout the summer. Preliminary indexing of peach seedlings with bud material taken from the affected trees in September, 1938, indicated that the condition was both bud-perpetuated and graft-transmissible.

Further inquiry disclosed the fact that, some years before, a number of trees in the block had been grafted with shoots of another Japanese variety, locally known as First, in order to provide for pollination of the Shiro bloom. Since signs of the disorder were found on almost every tree thus grafted and on a number of others besides, it was quite obvious that the virosis had been introduced into the Shiro block by means of the First grafts and that it had been spread by natural means. Accordingly, the scion material was traced to its source in another orchard, in which many of the First trees had foliar patterns so mild that they would readily escape observation, and in which a number of Shiro trees displayed typical symptoms (3). The only other known case of natural occurrence in the Niagara district was found near Beamsville on Italian prune, in combination with prune dwarf (10).

The disease has been called Shiro line-pattern mosaic (3) or Shiro plum line-pattern mosaic (7), but hereafter will be referred to as line pattern. The disease has been transmitted from Shiro to a number of plum, cherry, and peach varieties, and the present paper is concerned mainly with the symptomatology of line pattern on these several hosts. The relationship of the causal virus to some others affecting stone fruits is also discussed to a limited extent.

DIFFERENTIAL VARIETIES

The range of differential hosts employed in these experiments comprised the following species and varieties: *Prunus salicina* var. Shiro, Abundance, and Early Golden; *P. domestica* var. Italian prune, German prune, Imperial Gage, Reine Claude, Lombard, and Grand Duke; *P. cerasifera*, Myrobalan seedlings; *P. persica* var. Elberta, Rochester, and seedlings; *P. armeniaca* var. Niagara; *P. avium* var. Black Tartarian and Napoleon; *P. cerasus* var. Montmorency; and *P. mahaleb*, seedlings.

PROCEDURE

Both the double-budding technique and direct inoculation of orchard trees and nursery stock by budding have been employed. Since not only

[1] Contribution No. 821 from the Division of Botany and Plant Pathology, Science Service, Department of Agriculture, Ottawa, Canada.

the techniques but also their respective advantages and disadvantages have been discussed in recent articles (10, 11), there is no need for further comment here, except to say that the symptom sequences of line pattern, in contrast to those of tatter leaf (11), were not influenced appreciably by the mode of inoculation. Like prune dwarf (10), line pattern is very easily transmitted by budding, in fact, there have been no failures with any host at all susceptible to the virus, even when union between diseased bud and healthy stock was doubtful.

SYMPTOMATOLOGY

On the susceptible hosts, symptom expression of line pattern varied considerably in degree from year to year, being both more intense and more extensive for the variety when the weather remained cool for considerable periods in the early part of the growing season than when warm weather prevailed at that time. Some differences in pattern detail were also noted in different years. Almost invariably, symptoms appeared only on the leaves emerging in the spring. No distinctions could be drawn between the acute and chronic phases of the disease (11), except possibly on Montmorency, and, up to the present, there have been no indications that vigor was impaired to any appreciable extent.

On Shiro Plum.—The symptoms on Shiro were of two main types: yellow vein-banding (Fig. 1, E), and well-defined, brilliant, green-yellow patterns, usually of the oak-leaf type (Fig. 1, C), formed by single or multiple, irregular lines or bands. In early summer, the yellow of the patterns faded to a creamy white, which persisted for the remainder of the season. In most years, a regular sequence of pattern types could be traced, commencing with the oak-leaf and followed in successive leaves by a mixture of oak-leaf and vein-banding (Fig. 1, D), by over-all vein-banding and by vein-banding towards the leaf tips. New leaves emerging after early June were symptomless. In some years, one or other of the symptom types was more or less suppressed, depending upon conditions at leaf emergence.

On Abundance Plum.—Both in double-budding experiments and on an inoculated orchard tree, the symptoms on Abundance leaves somewhat resembled those on Shiro but were always much fainter and less numerous. Vein-banding was occasionally seen, but the predominant characteristic was the oak-leaf pattern, frequently in the typical form, but sometimes represented only by a narrow line either close to the margin near the leaf tip or on the serrations. On Abundance, the patterns were generally yellowish-green and more or less transient.

On Early Golden Plum.—No symptoms were observed in four years on Early Golden growing on a double-budded Myrobalan seedling, even though the Shiro shoot on the same stock showed striking symptoms and the Myrobalan shoot was moderately affected.

On Italian Prune.—When inoculated either by double-budding or directly on nursery stock, Italian prune either had inconspicuous green oak-leaf patterns or was symptomless, depending on whether or not the season was favor-

able for symptom expression. Line pattern was transmitted through artificially infected Italian prune to healthy Shiro without any apparent loss in virulence.

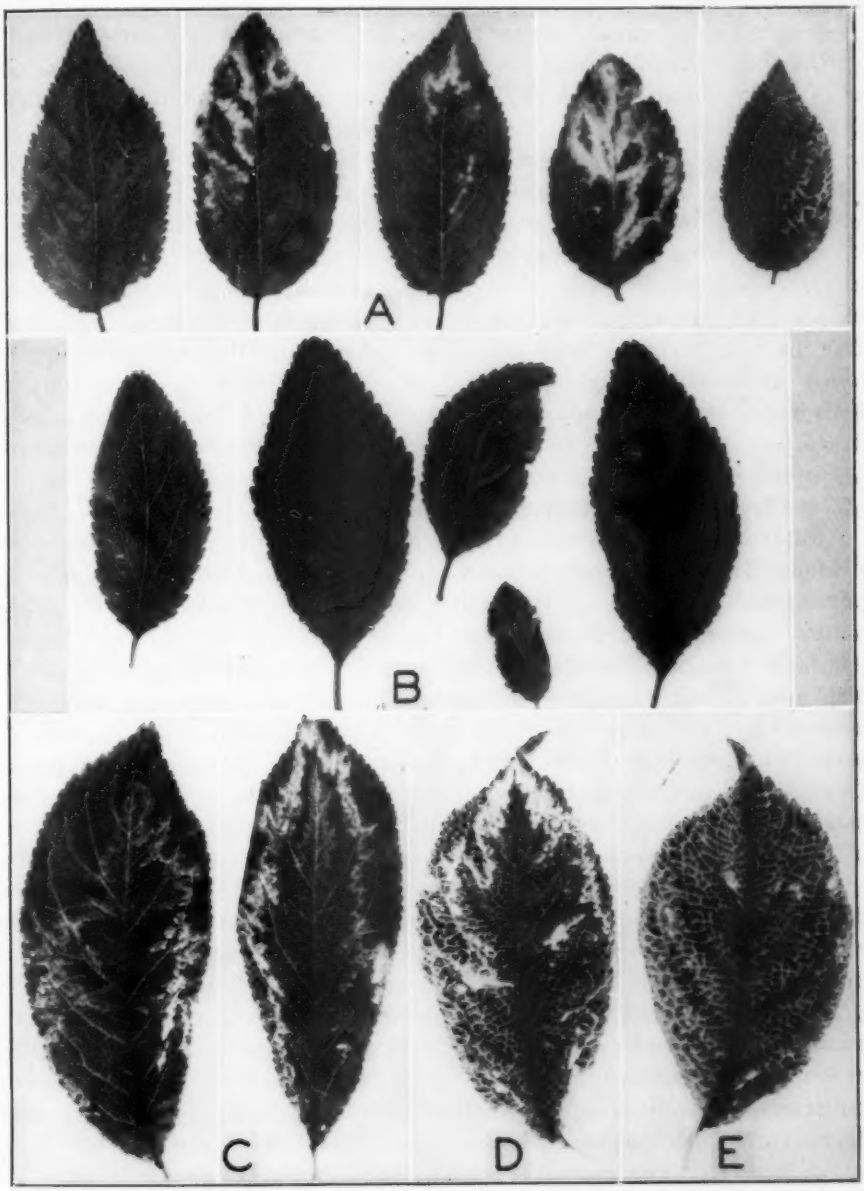

FIG. 1. Symptoms of line pattern on plums. A. Various markings on Myrobalan seedlings. B. Chlorotic and necrotic lines on Lombard. C, D, and E. Oak-leaf patterns, oak-leaf and vein-banding, and vein-banding, respectively, on Shiro.

On German Prune.—Inoculation by double-budding induced variously placed, fine, yellow lines or very irregular, spiky, oak-leaf patterns on the

early leaves of German prune. Symptoms on this variety were less vivid than those on Shiro, and also persisted throughout the season.

On Imperial Gage Plum.—One orchard tree, inoculated in September, 1938, has shown no symptoms since that time. The Shiro buds used for the purpose produced shoots having typical but comparatively mild symptoms.

On Reine Claude Plum.—Symptoms on Reine Claude, double-budded, were always mild, though slightly more conspicuous and numerous than those on Italian prune. Patterns were usually of the oak-leaf type, and were sometimes reduced to a narrow line near the tip of the leaf (Fig. 2, D). The markings were sometimes very slightly yellower than the normal leaf, but were usually best visible by transmitted light.

On Lombard Plum.—Early leaves near the point of inoculation, on nursery stock or trees, were occasionally and comparatively faintly marked with oak-leaf patterns, fairly large irregular rings, or vein-banding. More frequently and characteristically, a chlorotic line, which later became necrotic, outlined a triangular sector. The base of this sector lay at the leaf margin and the apex at or near the central vein (Fig. 1, B). Leaves marked in this fashion were usually distorted. In general, only a few leaves showed symptoms of any kind in any one year, and the disease seemed to spread slowly through individual trees.

On Grand Duke Plum.—No inoculations with line pattern alone were made on Grand Duke, but, in a series double-budded with the line pattern–prune dwarf mixture (10), leaves were conspicuously marked with fine lines, yellow oak-leaf, or large, irregular, yellow rings, which sometimes became partially necrotic. Since these patterns did not appear when Grand Duke was infected with prune dwarf alone, they are considered to have been caused by the line-pattern component of the mixture. The necrosis observed in Grand Duke leaves may have been due to the presence of the prune-dwarf virus, as in the Montmorency series described later. In the mixture, each virus appeared to aggravate the effects of the other on several varieties of the differential hosts used.

On Myrobalan Seedlings.—Individual seedlings of Myrobalan differed widely in their reaction to the line-pattern virus. At one extreme were the resistant or tolerant plants on which very few patterns were evident even in seasons most favorable for symptom expression. On these, symptoms, if they appeared at all, were usually a whitening of the tips of the marginal serrations and occasionally yellow-green oak-leaf patterns. At the other extreme were the susceptible seedlings on which the markings were usually indistinguishable from those on Shiro, in color, form, and distribution. Most Myrobalan plants, however, were only moderately susceptible. Some of the varied patterns to be seen on Myrobalan are illustrated in figure 1, A.

On Peaches.—Since the line-pattern symptoms expressed by the different peach varieties and seedlings varied little in quantity and less in kind, one general description will suffice. The most common symptom was a fine, irregular, wavering line on each side of the main vein of a leaf (Fig. 2, B),

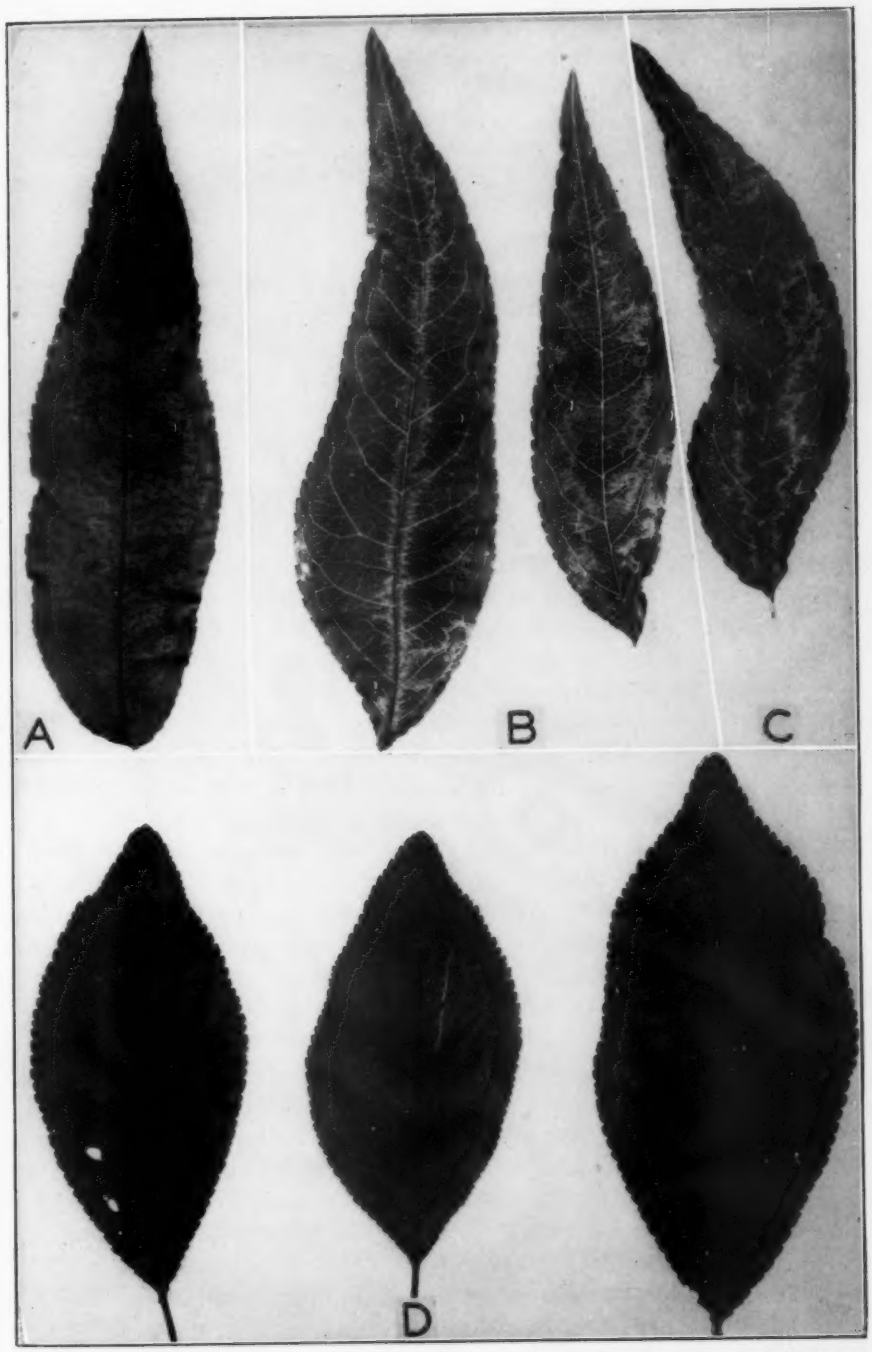

FIG. 2. Symptoms of line pattern on peach and plum. A, B, and C. Fine network, symmetrical lines, and broken lines, respectively, on peach. D. Faint oak-leaf and line patterns on Reine Claude, photographed by transmitted light.

placed anywhere between the mid-rib and the margin. The lines on a single leaf usually formed a symmetrical pattern but sometimes one was either broken or turned back on itself to form figures of various shapes (Fig. 2, C). Occasionally leaves were marked with a network of fine lines forming irregular patterns (Fig. 2, A), with fine, confluent rings, or with vein-banding. On the peach, patterns were mostly pale green, and tended to disappear later in the summer. The majority of leaves, especially those emerging later than early June, were symptomless. The number of leaves affected and sometimes the predominant symptom type varied from year to year according to growing conditions in the spring. In exceptionally favorable seasons, some yellow vein-banding and oak-leaf may appear.

On Apricot.—Up to the present time, no definite symptoms of line pattern have appeared on the apricot variety, Niagara, used in double-budding experiments, in spite of the fact that the disease was transmitted to the peach seedlings used for stock.

On Sweet Cherries.—The two varieties, Black Tartarian and Napoleon, responded in much the same way to inoculation with the line-pattern virus. Some of the symptoms resembled those induced by prune dwarf (10) or tatter leaf (11), for example, tiny, fine rings (Fig. 3, I), or larger, coarser rings and faint oak-leaf (Fig. 3, H). These patterns were pale green and tended to disappear in the course of the summer. Two symptoms, however, were peculiar to line pattern. The first was an inconspicuous line almost like a watermark, separating two areas of the leaf with slight differences in color. The second, and more common, was a series of yellow lines, varying from a hair's breadth to about 1 mm. These patterns were usually restricted to a small fraction, seldom more than a quarter, of the leaf surface. The finer lines frequently ran parallel to the secondary and some of the finer veins (Fig. 3, E), whereas the coarser ones delineated irregular patterns (Fig. 3, D). As on Shiro plum, the yellow markings persisted, turning creamy white later in the summer. The symptoms appeared in the vicinity of the inserted buds the first season but were more widely scattered in subsequent years. Most leaves were symptomless.

On Montmorency Cherry.—Symptoms on Montmorency nursery stock, in the first season after inoculation, were pale green markings in the form of broad bands, spots of various sizes, and coarse rings up to about 6 mm. in diameter (Fig. 3, A). The borders of these patterns were ill-defined and faded out into normal tissue. On a few leaves, a fairly coarse, irregular, yellow ring or partial ring appeared near the tip. A few, scattered, necrotic spots were observed at the bases of a number of affected leaves.

In June of the second year, leaves were marked with narrower and more sharply defined lines, variously placed with respect to the mid-rib (Fig. 3, B). These lines, though occasionally faintly tinged with yellow, were usually more like watermarks, more readily discernible by transmitted than by reflected light. Identically similar lines appeared at the same time on leaves of Montmorency inoculated with the prune dwarf–line pattern mix-

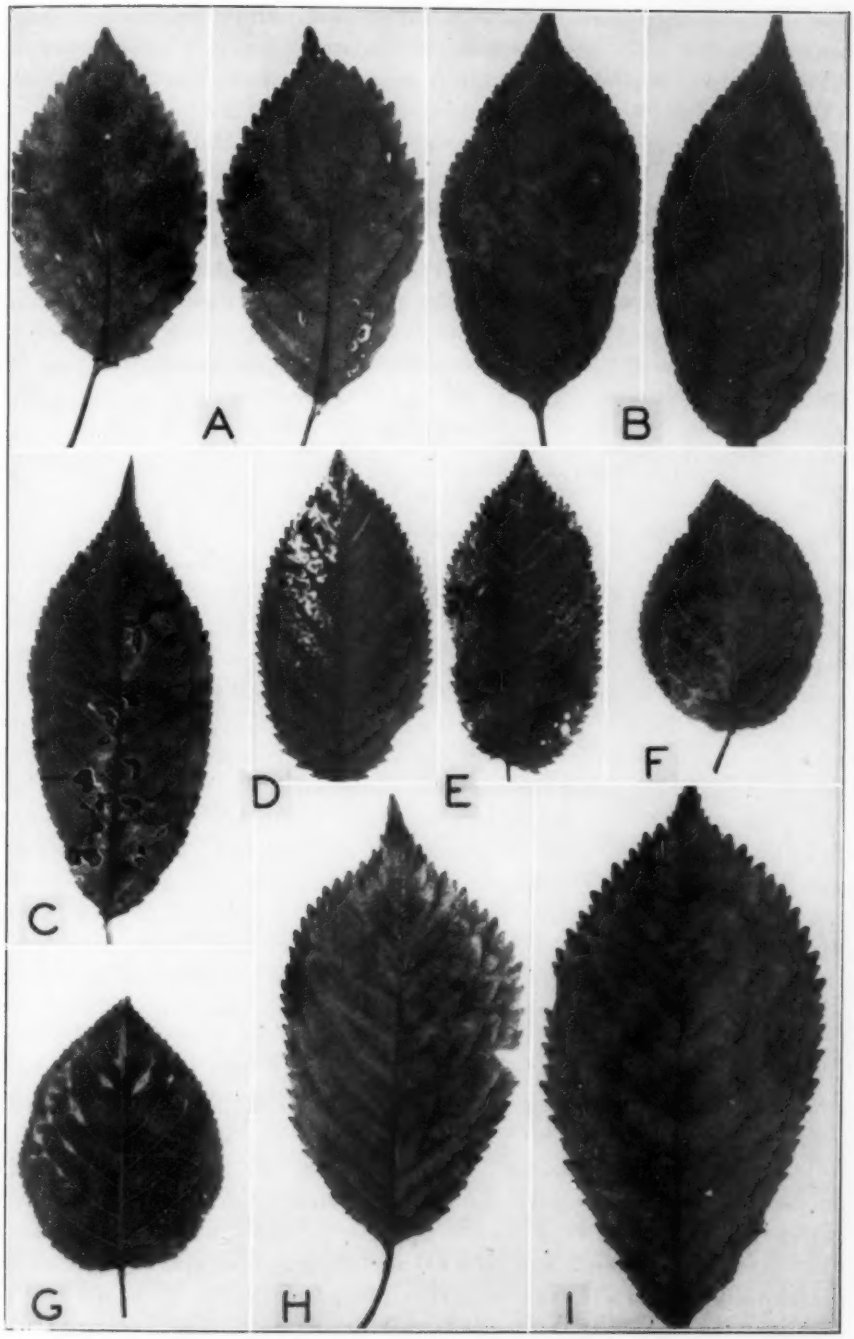

FIG. 3. Symptoms of line pattern on cherries. A and B. First and second season patterns, respectively, on Montmorency. C. Translucent lines and necrotic spotting on Montmorency affected with the line pattern–prune dwarf mixture. D. Coarse, yellow pattern, on Napoleon. E. Fine, yellow lines, on Black Tartarian. F and G. Partly modified oak-leaf, and short, yellow vein-bands, with necrosis of veins, respectively, on Mahaleb. H. Pale green lines, on Napoleon. I. Pale green rings, on Black Tartarian.

ture (10), but soon either went over into a dark, red-brown necrosis, or became associated with irregular necrotic spotting (Fig. 3, C). The necrotic phase did not appear with line pattern alone, and was evidently attributable to the combined effects of the two viruses.

On Mahaleb Seedlings.—In early spring, some Mahaleb leaves were marked either with oak-leaf patterns (Fig. 3, F) or with coarse rings and lines, sometimes darker and sometimes paler than normal. Of greater diagnostic value were the short, narrow, yellow bands which appeared along some of the veins of a number of leaves (Fig. 3, G) scattered over the plant. In these areas, the vein and, frequently, part of the yellow band eventually became necrotic, and either split or dropped out. In the early fall of the first season, a few of the newer leaves developed either coarse, pale, oak-leaf patterns or huge rings.

DISCUSSION

A comparison of the descriptions of the disease, as it appears on the different hosts, reveals a fundamental uniformity underlying the array of patterns, in so far as they can be referred to a linear type of variegation. Within the limits of that general type, however, considerable diversity of form was displayed, even on individual plants: oak-leaf, vein-banding, single and multiple lines and bands, and rings of various sizes. Although there was a tendency for one or other of these forms to predominate in a given species or variety of host, varietal reaction was largely a matter of intensity of expression. In this respect, there was a whole gamut of variation ranging from the barely perceptible patterns on First, Italian prune, and Reine Claude, through the green markings on peach and cherry leaves, to the brilliant yellow and cream figurations on Shiro and other hosts, and even to slight necroses on Mahaleb, Montmorency, and Lombard leaves. This suggests different levels of resistance and susceptibility on the part of the several host varieties. In this regard, the plum varieties of the three species, *Prunus domestica*, *P. salicina*, and *P. cerasifera*, were more variable than the peach and cherry varieties. It may be of interest here to note that varietal differences in susceptibility are not the same for line pattern as for either prune dwarf (10) or tatter leaf (11).

The intensity of symptoms varied not only with variety but with season. Observations over a six-year period lead to the conclusion that symptoms are suppressed in those leaves which develop when summer temperatures prevail. Though no accurate determinations of the temperature relations involved have been made, circumstantial evidence would indicate daily mean temperatures of 55° to 60° F. as critical for symptom expression. The possibility that other external and internal factors may also affect the virus-host relationship from year to year should not be overlooked.

Apart from growers' statements that crop yields of Shiro were apparently unaffected by line pattern, few data on that phase of the disease are yet available. However, fruits on infected Rochester and Elberta trees were normal in shape and quantity in 1944. Line pattern has also had little effect

upon vigor of growth to date, but whether it induces a gradual decline in vigor and crop production remains to be seen.

Present information would indicate that line pattern is not widely distributed in the Niagara district, since it has been found only in three orchards, and, although easily transmitted by grafting, has not yet turned up in masked form on plums indexed at the laboratory. Some evidence of natural dissemination in two of the affected orchards has been mentioned. Consequently, the existence of an insect vector may be postulated, but the infrequency of natural occurrence in the district and the absence of natural spread in the laboratory plantation during the last six years would suggest not only that the vector is comparatively rare but also that there is no transmission by sap transfer during pruning. The amply demonstrated possibility of inadvertent transmission of line pattern, as well as other viroses, through the use of scions in which the disease is more or less masked, emphasizes the necessity of great care in the selection of propagating material.

That some of the symptoms of line pattern on cherries and Mahaleb have much in common with some symptoms of tatter leaf and prune dwarf, indicates that there is some overlapping of the symptom expression of a number of distinct viroses on certain hosts. On the other hand, a number of characteristic symptoms, seasonal peculiarities, and differential host reactions serve not only to separate the line-pattern virus distinctively from other viruses under investigation here, but also to link it with viruses causing similar characteristic reactions elsewhere.

Through the courtesy of Donald Cation in supplying the necessary scions, the line-pattern virus that he worked with in Michigan (4) has been transferred to most of the differential hosts employed in Ontario. The symptoms of the Michigan virus on the respective differential hosts so resembled those induced by the native virus from Shiro that there is no reasonable doubt that the two viruses are very closely related, if not identical.

The virosis transferred by Valleau (9) from Abundance 422, Red June, and October Purple plums to peach (Valleau's figures 19, 22, and 23) as well as the one he found on Abundance in a nursery in 1931 (Valleau's figure 24) also appear very similar to those under observation in Ontario. Valleau's report that no patterns were seen on Shiro (9), leaves the identity of his virus still in question, and suggests either a difference in strain or the suppression of symptoms on Shiro under Kentucky conditions. The destruction of Japanese plums in Kentucky (7, p. 66), attributed by Valleau to ring spot or line pattern, is at variance with other reports on line pattern–like viroses, and may well have been caused by a mixture of viruses. Cation's experiments with plums from Kentucky (4) indicate that more than one virus was involved, and an increase in the virulence of line pattern when mixed with another virus has been noted.

Hildebrand (6) observed line pattern symptoms on Red June and Abundance in New York state, but his data are not sufficient to permit further comparison.

The symptoms on Santa Rosa plum of the "Vacaville plum mosaic," which Thomas and Rawlins (8) report as being similar to the Kentucky ring spot, appear to be of the line pattern type, but their description of the symptoms on peach as "consisting in mild mottling without any definite pattern" is not in agreement with the findings in Michigan (4) and Ontario. This again may be the result of differences either in virus strain or in environmental conditions. On the other hand, Thomas and Rawlins also refer to a second virosis on Santa Rosa and Duarte plums, symptoms of which on Myrobalan, according to description, seem to correspond well to those noted in Ontario on the same host. There is also a remarkable resemblance between the symptoms of banded chlorosis of Japanese cherries in Oregon, as described by Zeller and Milbrath (12), and those of line pattern on Shiro and Myrobalan in Ontario. It is, perhaps, more than coincidence that line pattern occurs in nature on Japanese varieties of plums in Kentucky, Michigan, New York, Ohio, and Ontario, that similar diseases were found on Japanese plums in California, and that banded chlorosis is common on Japanese cherries in Oregon.

References to line pattern-like virus diseases on stone fruits are also to be found in the European literature. G. and M. Arnaud (1) do not give any illustrations, but their general description of the symptoms of a mosaic on Myrobalan, apricot, and peach is equally applicable to the Ontario line-pattern virosis. On the other hand, the French virus differs from the Canadian one in that it can affect apricot but not Mahaleb. This would suggest strain differences in related viruses, though the different results with apricot could just as well be explained on the basis of varietal differences in the host.

Christoff's illustrations of narrow-striped variegation and ring-spot variegation (5) on Myrobalan and plum in Bulgaria show great similarity to the line-pattern symptoms on Myrobalan and Shiro. Furthermore, the symptoms of the Bulgarian disease on peach are described as light green to yellow thin lines and little rings. Atanasoff (2) also reported and illustrated a similar striped variegation on peach in Bulgaria. His virus induced yellow to white patterns on peach and distortion of fruit and in these respects differed from the Canadian one.

The viroses of the line pattern type, reported on stone fruits at widely separated points in North America and Europe, are then sufficiently alike in symptom expression to form a distinct group of diseases. Furthermore, some of them are reported as inducing symptoms only on leaves formed in the spring (1, 4, 5), and most do not greatly affect the vigor of the host (1, 4, 5, 8, 12). Since there is evidence that the causal viruses are not identical, it is suggested that a number of strains of the line-pattern virus are implicated. There also remains the possibility that some of the discrepancies may arise from contamination with other viruses, or from differences in environment.

SUMMARY

A striking yellow to white line-pattern virosis, found originally on Shiro

plum, has been transferred by budding to numerous species and varieties of the genus Prunus.

On all susceptible hosts, symptoms consisted of linear markings in diversified patterns, such as rings, lines, bands, oak-leaf, and vein-banding. These patterns occurred only on leaves produced in the spring, but persisted usually throughout the summer.

Symptoms varied in intensity and extent on the same host from year to year, being more plentiful when cool weather prevailed in the early part of the growing season.

Intensity of symptoms also varied greatly in plum varieties and Myrobalan seedlings. Patterns were virtually absent in Early Golden and Imperial Gage, barely perceptible and translucent in Italian prune, Reine Claude, and First, and brilliant yellow to white in Shiro and some Myrobalan seedlings. On Lombard, many of the patterns became necrotic even though comparatively few leaves per tree were affected.

On peaches, which did not show any wide differences in varietal susceptibility, symptoms usually took the form of pale green, irregular lines, rings, and vein-banding.

Faint, green rings and lines and yellowish to white patterns were found on the sweet cherry varieties, Black Tartarian and Napoleon. On the sour cherry variety, Montmorency, diffuse pale streaks, spots, and rings, and slight necrotic spotting appeared in the first, and narrow, translucent lines in the second season after inoculation. Mahaleb seedlings showed green rings and lines, as well as short, yellow, later necrotic, streaks along some of the main veins.

The writer wishes to express his sincere thanks and appreciation to Dr. G. H. Berkeley for much helpful advice and constructive criticism during the course of the investigation.

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TWO ALTERNARIA DISEASES OF CRUCIFEROUS PLANTS[1]

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An ever-increasing number of Alternaria species are being described on cruciferous plants. The two which have received the most attention are *Alternaria brassicae* (Berk.) Sacc. causing black leaf spot and *A. herculea* (Ell. & Mart.) Elliott, producing the grey leaf-spot disease. These two generally are present on mature plants, producing a browning of cauliflower and broccoli heads as well as a spotting of leaves and pods. These symptoms have been described in detail. The fact that the two organisms are carried on and in the seed and may cause serious loss by means of damping-off, a wire-stem, and cankers on the stems and cotyledons of seedlings has not been generally recognized in the literature. The present work was undertaken to verify former statements regarding pathogenicity, environmntal factors, and means of control, especially as they concern the production of plants in the seed-bed.

Alternaria herculea was once named *A. brassicae* (Berk.) Bolle,[3] and much confusion exists in the literature regarding the actual organism under discussion, since the writers often fail to give the author's name of the species to which they are referring. *A. brassicae* (Berk.) Sacc. and *A. herculea* (Ell. & Mart.) Elliott, however, can readily be distinguished by morphological and cultural characters.

Alternaria brassicae (Berk.) Sacc. is the species with small dark spores. It grows rapidly on potato-dextrose agar, forming colonies at first dark olive-green, then becoming almost black when the color of the conidia changes to brown. It sporulates abundantly and may produce long chains of more than ten conidia. It does not cause chromogenic reaction on rice medium. The spores germinate over a wide range of temperature, no sharp optimum temperature for germination being noticeable (Fig. 1).

Alternaria herculea (Ell. & Mart.) Elliott bears large light-brown spores. The cultures on potato-dextrose agar are white at first, then become brown when the conidia are formed, and on rice medium a light brown chromogenic reaction is produced. The fungus sporulates very poorly. The spores are sensitive to temperature variations; the optimum temperature for germination is somewhere between 17.2° and 21.1° C. Below and above these limits germination drops sharply (Fig. 2).

[1] An abridgement of a dissertation submitted to the Faculty of the Graduate School of Cornell University, August, 1944, as a major thesis in partial fulfillment of the requirements for the degree of Master of Science in Plant Pathology.

[2] Plant Pathologist in the Ministry of Agriculture, Brazil. Under a fellowship of the Institute of International Education, later with a prize of travel awarded by the Brazilian Government.

The author wishes to express his sincere appreciation to Drs. C. Chupp and L. J. Tyler for their helpful suggestions and criticisms.

[3] Bolle, P. C. Die durch Schwärzepilze (Phaeodictyae) erzeugten Pflanzenkrankheiten. Med. Phyt. Lab. Willie Commelin Scholten, Baarn. 7: 1–77. 1924.

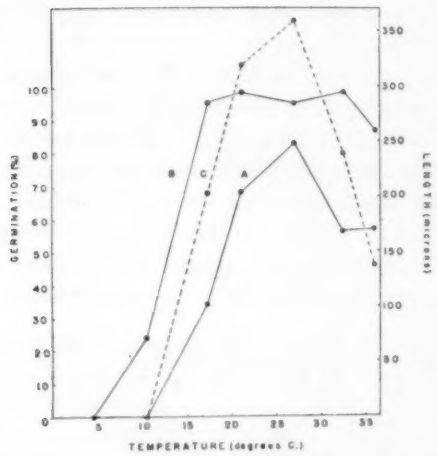

FIG. 1. Relation of temperature to rate of germination of conidia of *Alternaria brassicae* from a 17-day-old culture, after 9 hours (A), and 18 hours (B); and length of germ-tubes after 13 hours (C).

When flowers are inoculated with *Alternaria brassicae,* small, brown, ill-defined, and elongated lesions on the petals are produced. Minute, circular, brown spots appear on the flower pedicels and calyces. On the inoculated pods the lesions are small, purple-brown on green pods, or dark brown on dry pods. The young pods may be distorted, cease to grow, and the affected pods dry out from the tip and shatter prematurely. The seeds themselves do not have definite lesions, but seeds in infected young pods do not develop fully, and those in infected, maturing pods are shriveled and non-viable.

The cotyledons of seedlings developed from infested or infected seeds exhibit sharply delimited dark-brown to black spots, usually circular, and more or less sunken (Fig. 3, a). These spots appear more frequently on the

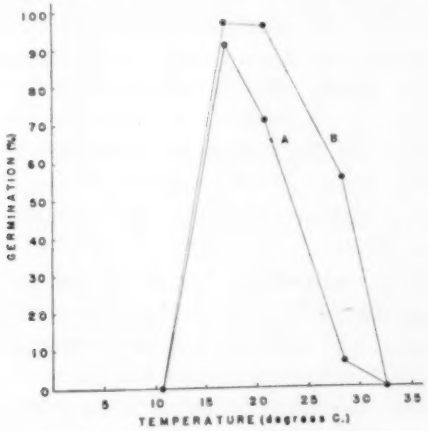

FIG. 2. Relation of temperature to rate of germination of conidia of *Alternaria herculea* from a 20-day-old culture, after 8 hours (A), and 13 hours (B).

dorsal side and their location seems to be related to the folding of the cotyledons in the seed (Fig. 3).

Damping-off may occur when very young seedlings are affected. The spots on the hypocotyl of young seedlings are narrow, dark-brown to black and about one millimeter long (Fig. 3, b). The base of the stem has a brown, soft, hydrotic area, and later the plant bends over and dies. When infection occurs after the tissues have become harder, the seedlings are not killed, but the cortical cells at the base of the stem collapse, forming a dark, thin layer of dead cells. Because of this withering the diameter of the stem below the cotyledonal leaf scars is reduced. As the central cylinder is not affected, the

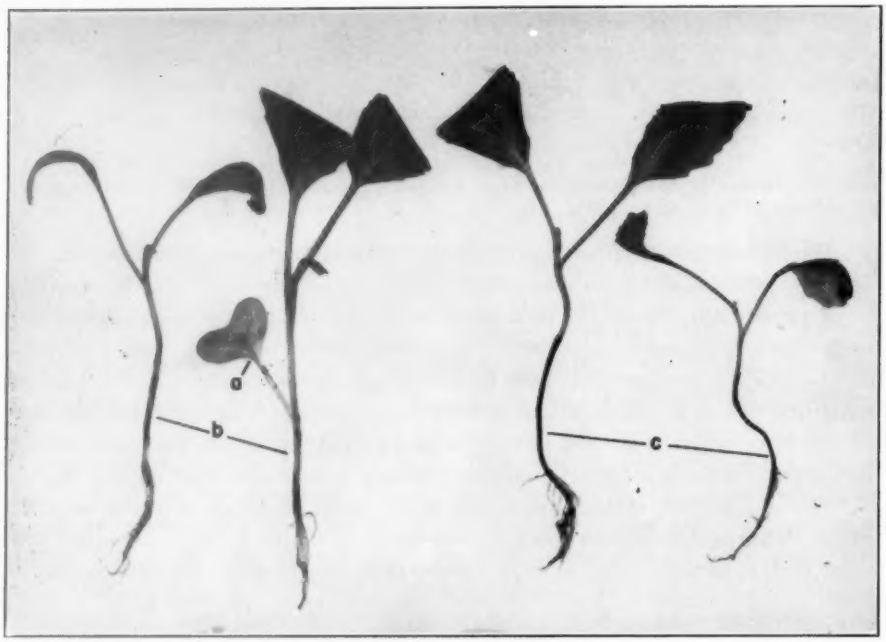

FIG. 3. Spotting of the cotyledon (a), and two phases of the damping-off stage (b and c) on seedlings of cabbage grown from inoculated seed sown in steam-sterilized soil.

plants can survive, but the stems become distorted and the growth of the plant is impaired (Fig. 3, c). Such symptoms are similar to those of wire-stem caused by *Rhizoctonia solani* Kühn. Pre-emergence damping-off may occur.

When surface-inoculated cabbage seeds were sown in sterilized soil in greenhouse flats there was 48.3 per cent of pre-emergence damping-off; and of the plants remaining, 51.7 per cent had post-emergence damping-off, and 38.4 per cent had wire-stem within twenty-five days.

Inoculation experiments were conducted in the greenhouse for the studies on pathogenicity. Conidia of *Alternaria brassicae* produced on potato-dextrose agar were suspended in 1–500 gelatin solution and atomized on the

plants, which were then placed in a moist chamber for approximately 48 hours. Plants then were transferred to greenhouse benches. The gelatin was used to improve the wetting ability of the spore suspension, resulting in a more uniform deposition of the inoculum, and it eliminated the necessity of rubbing the leaves, as practiced by previous workers.

The inoculation experiments demonstrated that *Alternaria brassicae* (as well as *A. herculea*) is a virulent pathogen and that injury is not needed for infection. The somewhat general belief that the fungus attacks preferably the older leaves seems not to be sound. A better explanation for the fact that mostly the lower leaves (which are the older) are infected is that the organism sporulates on decaying parts in contact with the soil and the spores are splashed by the rain drops over the lower leaves. Sometimes these leaves remain in contact with the soil and are likely to become infected because of the proximity of conidia formed on the debris.

The minimum period of wetting required for leaf infection was also investigated. Inoculated cabbage plants were incubated in a moist chamber and groups of them were removed at intervals of six hours. The temperatures ranged from 19° to 28° C. Infection was not evident in plants removed after 6, 12, and 18 hours, whereas those remaining in the moist chamber for 24 hours or longer were infected.

Examination of several seed samples revealed that frequently spores are carried on the surface of the seed and that these spores may remain viable for a long time. Spores obtained from seed samples about twenty months old and stored at 32° F. for fourteen months showed high germinability. When planted on plain agar several of the seeds were covered with mycelium and spores, and the growth of the seedlings attacked by the fungus was checked. When seeds were sown in pots, characteristic symptoms of the disease were produced on the seedlings.

There is good evidence that the fungus sometimes is carried as latent mycelium in the seed. Growth of *Alternaria brassicae* developed from naturally infected seeds of kale subjected to each of the following treatments: (a) hot water at 52° C. for 20 minutes; (b) seeds dipped in 95 per cent alcohol, treated with corrosive sublimate 1–1000 for 5 minutes, and washed with four changes of sterilized distilled water; (c) seeds dipped in 95 per cent alcohol, treated with corrosive sublimate 1–1000 for 5 minutes, and washed with two changes of Clorox 25 per cent; (d) seeds treated with Clorox 25 per cent for 5 minutes and dried on filter paper.

Tests were made to check the effect of hot water and of corrosive sublimate on spores. Spores of 12-day-old cultures suspended in water were held at 52° C. for 5 minutes in a water bath. In another experiment, spores of a 12-day-old culture were suspended in corrosive sublimate 1–1000 for 3 minutes, caught on filter paper, and then washed with four changes of sterilized distilled water. These treated spores when placed in 1 per cent sugar solution did not germinate within 24 hours, whereas 97 per cent of the untreated spores germinated.

These facts lead to the conclusion that a latent mycelium must be present inside the seeds, as it does not seem possible that the spores on the surface of the seed could stand the treatments that were given.

In other experiments, seeds of bok-toy and radish were dipped in 95 per cent alcohol, treated with corrosive sublimate 1–1000 for 5 minutes, washed with three changes of sterilized distilled water and planted on potato-dextrose agar. Within five days a white mycelium grew from a few seeds, and spores of *Alternaria herculea* were found in microscopic mounts from these cultures. A hyaline mycelium was present in the epidermis of seeds of the same samples. Sections of radish seeds contained a hyaline, septate, branched mycelium, 4 microns wide, that extended into the endosperm layer. These observations support the statement that this pathogen can be carried underneath the seed coat.

Since *Alternaria brassicae* sporulates abundantly on decaying vegetable matter, the soil may be an important harborer of the pathogen. Thus, the pathogen can possibly be carried from the seedling beds to the field in soil adhering to the seedling roots, or on seedlings that have been splashed by rain or overhead irrigation. It also is possible for the seedling to have infections in an incipient stage that later result in the killing of the plant or part of it, and consequent production of spores in the field. In this manner, the seedlings grown in infested seedling beds may be of great importance in the dissemination of the inoculum.

The longevity of spores was tested by exposing cultures of the fungus on potato-dextrose agar to outdoor weather during January to July, in which the organism was exposed to temperatures ranging from $-23°$ to $+30°$ C. At the end of this period the spores were still viable and pathogenic, as shown by their germination on slides and their ability to induce disease in inoculated cabbage plants. These results, however, do not warrant final conclusions regarding the behavior of the organism in the soil.

The effectiveness of chemicals in seed treatment for the control of *Alternaria brassicae* was studied in the greenhouse. For these studies, Alternaria-free cabbage seed was inoculated with spores suspended in a gelatin solution (1–500), dried, and treated. The dosages of the chemical materials applied as dusts were dependent upon the amount of chemical adhering to the seeds. Aside from the dust treatments, some seeds were soaked in a 1–1000 solution of corrosive sublimate for 5 minutes, and dried. Inoculated and non-inoculated untreated seeds were included to check the germination of the seeds. Four replications of 100 seeds for each treatment were planted in flats in steam-sterilized soil. Pre-emergence damping-off was estimated by assuming that the differences in stand between the non-inoculated check and the treatments were due to the pathogen. Table 1 gives the total number of seedlings affected for each treatment, after 25 days.

Semesan and Arasan were effective in reducing the amount of damping-off and wire-stem. While Wettable Spergon checked the pre-emergence damping-off, it was not efficient against post-emergence damping-off. The

TABLE 1.—*Results of seed treatments for the control of Alternaria brassicae on surface-inoculated cabbage seeds*

Treatment	Total emergence	Final stand	Damping-off		Wire stem[b]
			Pre-emergence (estimate)	Post-emergence[a]	
Arasan	283	278	9	5	4
Semesan	289	285	3	4	9
Fermate	273	247	19	26	23
Wettable 604	272	269	20	3	6
Wettable Spergon	284	263	8	21	5
Corrosive sublimate	269	218	23	51	13
Check (inoculated)	151	73	141	78	28
Check (not inoculated)	292	290	0	0	0

[a] Post-emergence damping-off:
Least difference for odds of 19:1 = 31.5 seedlings
" " " " " 99:1 = 43.2 seedlings

[b] Wire stem:
Least difference for odds of 19:1 = 14.5 seedlings
" " " " " 99:1 = 19.9 seedlings

reverse was shown by Wettable 604. The other two treatments did not control damping-off.

SUMMARY

Alternaria brassicae (Berk.) Sacc. that causes a leaf spot, pod spot, and general browning of heads of cauliflower and broccoli and other cruciferous plants is also the cause of damping-off, wire-stem, and spotting of seedlings. This pathogen is readily distinguished by morphological and cultural characters from *A. herculea* (Ell. & Mart.) Elliott which likewise causes a disease of cruciferous plants.

Both Alternaria species are virulent pathogens and are able to infect the suscept at any age and independently of any injury. A period of wetting for at least 18 hours is necessary for infection.

The seeds may carry both pathogens as spores or as latent mycelium in the seed.

The spores of *Alternaria brassicae* may retain their viability and their pathogenicity for more than six months. Seedlings grown in infested seed beds may carry the inoculum to the field. In the field the sources of inoculum are the dead lesions and the decaying plant parts on the soil. Water is the main agent of dissemination.

Semesan and Arasan were effective in reducing the amount of damping-off and wire-stem on seedlings originated from surface inoculated seeds.

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INOCULATION EXPERIMENTS WITH PSEUDOMONAS RIBICOLA

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INTRODUCTION

The first pathogenicity test of *Psudomonas ribicola,* which causes a defoliating spot disease of *Ribes aureum* Pursh. described by Bohn and Maloit (1), was made in the field at Cheyenne, Wyoming, in July, 1940. Both surfaces of leaves on vigorous shoots were sprayed with $\frac{1}{10}$ dilutions of 48-hour beef-extract broth cultures before, during, and immediately after a thundershower between 6 p.m. and 8 p.m. Each shoot in 1 series was wrapped in moistened cheesecloth immediately after spraying; each shoot in a second series was enclosed in a large, brown paper bag. In spite of these seemingly excellent conditions for infection, no leaf spots occurred on the 400 or more leaves on 24 shoots inoculated. Because the 6 bacterial isolates used were typical of numerous isolates obtained by approved methods, and because bacteria were found in all spots of the type under study that were examined with the microscope in 1939 and 1940, other methods of inoculation were tried.

In January, 1941, thirty plants in 2-gallon stone jars were pre-treated for 24 hours in a greenhouse moist chamber. Three check plants were sprayed with sterile broth and 1 young leaf on each plant was pricked slightly 10 to 12 times with a sterilized needle immediately after spraying. Each remaining lot of 3 plants was inoculated with 1 of 9 different bacterial isolates. Each plant was replaced in the moist chamber immediately after inoculation. No spots occurred on the checks; twelve spots resulted from the spray inoculations (approximately 600 leaves); 31 spots, from the needle punctures (approximately 300 punctures). The few infected spots that resulted from spray application of bacterial suspensions may have occurred in wounds caused by insects although very few insects (white flies) were present on these plants. The low proportion of infected spots resulting from needle punctures and the occurrence of holes in the centers of the spots suggested that some other method causing smaller wounds might yield infection spots more abundant and more comparable with those resulting from natural infection.

LITERATURE

A review of the general literature revealed a paucity of methods of inoculation recommended for bacterial plant pathogens. This is especially true with diseases that do not become systemic, infections being limited to local lesions on leaves, tender stems, and fruits (2, 3, 6, 7, 11, 12, 13, 14). The

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methods generally recommended are based on modes of ingress of bacteria into plant tissues in natural infections. Spraying has been recommended for those bacteria that gain access to susceptible tissues through stomata or directly through hydathodes, lenticels, nectaries, or other noncutinized surfaces. Treatment of host plants in moist chambers before and after spraying has been recommended to keep the stomata open, and to increase moisture present in intercellular spaces and on outer surfaces. Forced spraying with water before application of the bacterial suspension and forced spraying with bacterial suspensions have been used to aid bacteria to penetrate stomatal openings. Wounding with a needle or similar tool has been recommended for those bacteria that gain access to susceptible tissues through wounds caused by insects, hail, or other agents. It has been recommended that with any particular disease under study, inoculation methods should imitate natural infection methods under comparable conditions of the host and environment. Therefore, observations on natural infections of plants in the field were reviewed.

NATURAL INFECTION OF GOLDEN CURRANT BY PSEUDOMONAS RIBICOLA

At Cheyenne, Wyoming, bacterial spots appear on currants in the middle of June. The spots are most abundant on the first leaves to grow in the spring and the general absence of distortion suggests that infection occurs after the leaves have nearly matured. Therefore, natural infection must occur in late May or early June. The weather at Cheyenne during this part of the spring is cool, cloudy, and windy. The maximum daily temperature usually is between 65° and 80° F. (19° to 26° C.); the minimum, between 35° and 50° F. (1° to 10° C.). Although total rainfall is slight, the sky usually is cloudy, the relative humidity is high on frequent occasions, and thundershowers, often accompanied by hail or sleet, are frequent. The average wind velocity for May and June usually is 9 to 12 miles an hour, and high winds or gales frequently occur. These observations on natural infection and the weather data suggest that natural infection by *Pseudomonas ribicola* in currants occurs in minute wounds resulting from wind-blown hail, sleet, or sand or from buffeting of leaves against other leaves and branches. The observations suggest also that infection is favored by low temperature and high relative humidity. These conclusions are supported by the occurrence of necrotic bacterial lesions in hail-injured spots on young shoots and by the continued enlargement of spots in detached leaves placed in a moist chamber in the refrigerator at approximately 3° C.

COMPARISON OF INOCULATION TECHNIQUES WITH BACTERIAL SPOT OF CURRANT

Three techniques of inoculation that might yield results with currants more comparable to natural infection than do those usually recommended for use with bacteria were suggested by the virus infection techniques described by Holmes (4), Jones (5), Rawlins and Tompkins (8, 9, 10), and

Riker, Ark, Elliot, Hildebrand, and Leach (12). These methods are (a) Bacterial suspensions from agar cultures can be stippled on leaves supported by fingers or wooden pot labels with previously disinfected (with 70 per cent alcohol) lacquer brushes that have short, moderately stiff bristles; (b) Bacterial suspensions can be rubbed on leaves with previously sterilized fingers (2) or cotton or cheesecloth swabs (4); (c) Bacterial suspensions can be rubbed on leaves (as in b) dusted with carborundum (8, 9).

These methods were compared with spraying and needle-wounding methods using *Pseudomonas ribicola* on leaves of *Ribes aureum* grown in pots in the greenhouse during the winter of 1940–41. As all of the repeated experiments yielded similar results, only one, which included all of the methods compared at this time, is reported in detail.

Two-year-old currant seedlings were transplanted from the field nursery to 3-inch pots and forced into growth in the greenhouse. Vigorous seedlings were selected for inoculation. Following inoculation, the plants, with the exception of 1 group dusted with carborundum, were placed under bell jars in the laboratory at approximately 21° C. for 48 hours, then incubated in a cool greenhouse (day 18° to 25° C., night 8° to 15° C.) for 2 weeks. Tap water suspensions of *Pseudomonas ribicola* from 4-day-old beef-extract-dextrose-agar cultures were used as inoculum. Instruments and hands were disinfected in 70 per cent alcohol and then washed in tap water between treatments. Cotton, cloth, and pot labels were heated in glass containers in an electric oven at 170° to 180° C. for 2 hours before use. Five treatments were compared.

1. Suspensions were brushed with lacquer brushes on several leaves of each of 5 plants. Check leaves were brushed with tap water.

2. Suspensions were sprayed on the leaves of 5 plants. Check plants were sprayed with water.

3. Suspensions were rubbed on several leaves of each of 5 plants with coarse cheesecloth swabs. Check leaves were rubbed with a swab wet with water.

4. Suspensions were rubbed on several leaves of each of 5 plants with coarse cheesecloth swabs after the leaves had been dusted with 300-mesh carborundum. Check leaves were dusted with carborundum and rubbed with a swab wet with water.

5. Three plants were inoculated as in treatment 4, but left uncovered in the greenhouse immediately after inoculation (*i.e.*, they were not put in a moist chamber).

After 2 weeks' incubation numerous typical bacterial spots (15 to 50 on a leaf) had developed on leaves inoculated by brushing (Treatment 1) and on those inoculated by rubbing carborundum-dusted leaves (Treatments 4 and 5); no difference could be noticed between carborundum-dusted inoculated plants incubated 48 hours under bell jars in the laboratory and those left uncovered in the greenhouse for the entire incubation period. Fewer spots (2 to 13 on a leaf) had developed on leaves rubbed without carborundum (Treatment 3), and only 1 spot occurred on the sprayed leaves.

No effects of brushing check leaves could be seen with the unaided eye; but slightly raised streaks, not different in color from the rest of the leaf, were visible at 10 × magnification or greater. It is possible that injury was limited to removal of cuticle, but some epidermal cells may have been injured. This type of injury probably occurs commonly in the field at Cheyenne as leaves are brushed against each other and against stems by the wind. A few small brown flecks, barely visible to the unaided eye, occurred on carborundum-dusted leaves rubbed with a cheesecloth swab. Injury not visible to the unaided eye obviously occurred also (9). This type of injury would be expected to occur commonly at Cheyenne from wind-driven sand and sleet. No injury was observed on leaves sprayed or on those rubbed without carborundum.

Additional inoculation experiments were conducted during the winter of 1943–44. In two series of tests, water suspensions of *Pseudomonas ribicola* from potato-beef-extract-sucrose-agar cultures were used on young leaves of *Ribes aureum* clones grown in 2-gallon stone jars in the greenhouse. The plants were incubated in a greenhouse moist chamber at 15° to 25° C. for 24 hours after inoculation; then removed to a cool greenhouse (18° to 25° C. day and 8° to 15° C. night) for incubation. All implements were disinfected in 70 per cent alcohol or by heat before use.

Three treatments were compared in the first series.

1. The suspension was sprayed on both surfaces of the leaves of 4 plants; checks were sprayed with sterile water.

2. The leaves of 4 plants were dusted with 300-mesh carborundum and sprayed with the suspension; checks were dusted and sprayed with sterile water.

3. The leaves of 10 plants were dusted with 300-mesh carborundum and rubbed with a cotton swab; checks were dusted and swabbed with a sterile moist pad.

No injury or infection resulted from the simple spray treatment. All leaves dusted with carborundum and sprayed with bacterial suspensions developed moderate numbers (2 to 15) of typical bacterial spots; checks showed no injury. All leaves dusted with carborundum and rubbed with a cotton swab dipped in bacterial suspensions developed numerous (30 to 80) typical spots. Check leaves dusted with carborundum and rubbed with sterile pads developed a few minute brown flecks which apparently resulted from mechanical injury.

In the second series of tests, 7 treatments were compared on currant plants kept in a greenhouse moist chamber for 15 hours before and 24 hours after treatment and incubated for two weeks in a cool greenhouse.

1. A moderately turbid bacterial suspension was sprayed on both surfaces of the leaves. Checks were sprayed with sterile water.

2. The bacterial suspension, with 300-mesh carborundum added, was sprayed on both surfaces of the leaves. Checks were sprayed with a sterile carborundum suspension.

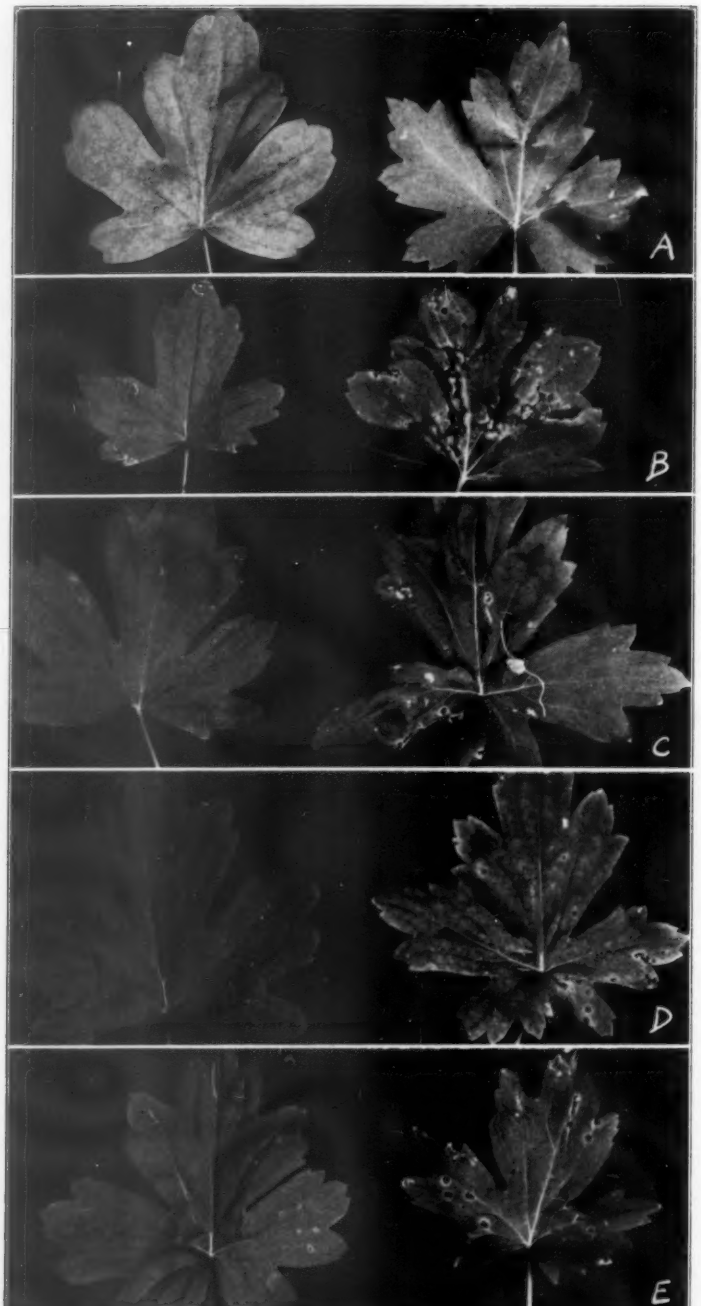

FIG. 1. Leaves of *Ribes aureum* inoculated with suspensions of *Pseudomonas ribicola*. Greenhouse, Cheyenne, Wyoming, 1944. Checks at left, inoculated leaves at right. A, Spray; B, Forced spray; C, Cotton swab; D, Carborundum dust and cotton swab; E, Needle.

3. A $\frac{1}{10}$ dilution of the suspension used in treatment 1 was sprayed against leaves with sufficient force to watersoak small regions of leaf tissue. Checks were sprayed similarly with sterile water.

4. Needles were dipped in a moistened agar culture and used to stipple leaves. Checks were stippled with sterile needles.

5. Needles were dipped in a moistened agar culture and drawn across leaves with light pressure insufficient to cause obvious wounds. Checks were similarly scratched with sterile needles.

6. Leaves were rubbed with a cotton pad moistened with the bacterial suspension used in treatment 1. Check leaves were rubbed with a cotton pad moistened with sterile water.

TABLE 1.—*Effectiveness of different methods of inoculating leaves of golden currant with Pseudomonas ribicola in the greenhouse at Cheyenne, Wyoming, March, 1944*

Method	Plants treated, excepting checks	Spots on		Visible injury on checks	Comparison with natural infection
		Young leaves	Mature leaves		
	Number				
1. Spray	3	Rare	None	None	Spots typical
2. Spray, carborundum	3	Moderate	Rare	do	do
3. Forced spray	3	Abundant	Abundant	do	Severe killing of large areas and extreme distortion rarely seen in natural infections
4. Needle puncture	3	On 75 per cent of punctures	On 10 per cent of punctures	Holes	Similar holes rare in spots resulting from natural infections
5. Needle scratch	3	On 75 per cent of scratches	On 10 per cent of scratches	Scratches, distortion	Similar mechanical injury not abundant in natural infections
6. Cotton swab	5	Moderate	None	None	Spots typical
7. Carborundum dust and cotton swab	5	Abundant	Rare	Slight flecks	Spots typical. Comparable flecks would probably be overlooked in natural infections

7. Leaves were dusted with 300-mesh carborundum and rubbed with a cotton pad moistened with the bacterial suspension used in treatment 1. Check leaves were dusted with carborundum and rubbed with a cotton pad moistened with sterile water.

The data on infection and mechanical injury, together with comments on comparisons with natural infections are presented in table 1 and figure 1. It is apparent that *Pseudomonas ribicola* is a wound parasite and rarely enters currant leaves through stomata. The best of these seven techniques of inoculation was that in which leaves were dusted with carborundum and rubbed with a pad moistened with a suspension of the bacteria. Other methods either resulted in few foci of infection or caused injury not comparable to injury observed in naturally infected currants in the field.

DISCUSSION

The abundant infections that resulted from applications of suspensions of *Pseudomonas ribicola* to wounds in leaves of *Ribes aureum* demonstrated that this bacterium is the pathogen associated with a necrotic spot disease of the golden currant. The general failure to obtain infections in leaves on plants kept in moist chambers and sprayed with suspensions of this organism indicated that the bacterium is a wound parasite and usually does not enter leaves through stomata. Observations on the abundance and appearance of infections that resulted from applications of suspensions by different methods, together with observations on natural infections and the weather, indicated that *Ps. ribicola* is a wound parasite which usually enters currants through minute wounds caused by wind-driven sand and sleet and buffeting of plant organs against each other in the wind.

The abundant typical infections by *Pseudomonas ribicola* in currant leaves obtained with 2 inoculation techniques, new or little described for use with bacterial plant pathogens, suggest that these methods may have value in studies on other bacterial diseases of plants. The techniques are (1) application of bacterial suspensions with lacquer brushes or other kinds that have moderately stiff bristles, and (2) application of bacterial suspensions with cotton or cloth pads to leaves or other organs dusted with carborundum (Rawlins and Tompkins' carborundum technique for virus inoculation). These techniques have other characteristics that suggest their probable utility in plant breeding programs concerned with resistance to diseases caused by bacteria and fungi. Inoculations can be made with comparative ease and rapidity and the symptoms, at least those of bacterial spot of currant, are comparable to symptoms that result from natural infections. Numerous infections result when these techniques are used on plants placed on open greenhouse benches, *i.e.*, without the use of moist chambers. These results suggest that ingress of bacteria into susceptible tissues occurs at the time of inoculation. This agrees with the observations by Holmes (4) on pad inoculations with viruliferous plant extracts. If this is true, the brush and the carborundum techniques may have value in large-scale inoculation experiments with plant pathogenic bacteria and fungi, especially those dependent on wounds for ingress. They should be particularly useful with hosts that have hard, thickly cutinized leaves, and in field inoculations where control of relative humidity is difficult.

A sample of 600-mesh carborundum was unsatisfactory in these experiments, while a sample of 300-mesh carborundum yielded excellent results. Microscopic examination revealed few angular particles in the 600-mesh sample; most of the particles were smooth and nearly spherical. The 300-mesh sample consisted nearly entirely of angular, sharp-edged particles with dimensions varying from 5 to 20 microns. These data and the discussion of carborundum samples by Rawlins and Tompkins (10) indicate that one should be able to predict the value of a sample for use in inoculation work

by microscopical examination. Although 300-mesh dust was used in all experiments reported here, other sizes may be found more satisfactory for use with hosts other than currant.

The atypical symptoms caused by watersoaking leaf tissue with bacterial suspensions was further indicated in experiments with other plants. Regions of leaves of tomato, raspberry, and strawberry watersoaked by forced spraying with suspensions of *Pseudomonas ribicola* were killed within 3 to 5 days although sterile water similarly forced into them caused little or no injury. Repeated inoculations by other methods, including the carborundum dust and cotton pad technique, failed to yield infection in these plants. Apparently, necrosis following forced spray applications severe enough to cause saturation of leaf tissues with bacterial suspensions is not a reliable test of pathogenicity. This does not apply, of course, to the spray method in which leaves are watersoaked with sterile water before application of bacterial suspensions to the surface. Leaf tissues usually are little injured by watersoaking with sterile water, although occasionally injury has occurred in tomato leaves so treated. The results obtained with forced sprays on immune plants indicate that the use of sterile water as a check for inoculations with bacterial suspensions may not be a reliable check. It would seem advisable to use suspensions of killed bacteria or suspensions of other species of bacteria not pathogenic to the host as checks in critical studies on parasitism.

SUMMARY

Methods of inoculation generally recommended for use with bacterial plant pathogens resulted in little infection by *Pseudomonas ribicola* in currant leaves or in symptoms unlike those resulting from natural infections. Inoculations with moderately stiff brushes and with cotton pads rubbed on leaves dusted with 300-mesh carborundum yielded abundant infections which produced symptoms comparable to those resulting from natural infections. Intermediate numbers of infections resulted from spray applications of bacterial suspensions on carborundum-dusted leaves. These results, together with observations on natural infections and the weather, indicated that *Pseudomonas ribicola* is a wound parasite which naturally enters currant leaves through minute wounds caused by buffeting of leaves against other leaves and branches in the wind and by wind-driven sand and sleet.

The probable value of these techniques in programs on breeding crop plants for disease-resistance is suggested. The brush and the carborundum dust and cotton pad techniques should prove successful with bacteria that usually invade hosts through minute wounds, especially with hosts that have hard, thickly cutinized surfaces.

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INFECTION EXPERIMENTS WITH DETACHED WATER-CONGESTED LEAVES[1]

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The relation of water congestion (4) in plant tissues to disease predisposition and to varietal susceptibility is being investigated through a number of different means in this laboratory. One of the more simple and promising methods of study has involved the use of detached leaves of plants treated in various manners before, during, and after inoculation with a variety of parasitic and saprophytic organisms, viruses, toxins, chemicals, and inert substances such as India ink and dyes. The use of detached leaves for infection experiments is not new, but in combination with prior water congestion introduces additional highly favorable conditions for infection. The detached-leaf method permits ready comparisons of tests under a fairly uniform set of conditions which are not otherwise easily obtained or maintained. A number of tests, for example, may be conducted on a single leaf when using such large leaves as those of tobacco. Only a brief summary of the results of these studies need be presented to suggest its applicability to further studies and to show the important relationship of water congestion in leaf tissues to the mechanism of infection and the further development of disease. Earlier papers from this laboratory have dealt with some of the more general aspects of the subject (3). The problem is related to, but not identical with, the more frequently recognized phenomenon of "storm water-soaking" as described by Clayton (1).

MATERIALS AND METHODS

Detached, disease-free leaves of many plants, including tobacco, may be maintained in approximately normal condition in covered containers for periods of 6 to 12 days at room temperatures. Using sterilized refrigerator pans ($12 \times 7 \times 4$ inches) for large individual leaves and for different treatments, fairly satisfactory conditions for infection on such living tissue cultures may be obtained. The absence of light and the high humidity provided by moistened, paper toweling on the bottom of the pan seem to favor the prolongation of the life of the detached leaf while at the same time providing suitable conditions for disease development. Removing the covers momentarily 2 or 3 times daily supplies sufficient aeration. Water-congested leaves decline faster than uncongested leaves, but the congestion need not be allowed to continue for more than 18–24 hours at the maximum. Tobacco leaves appear to be well adapted to this technique; whereas potato leaves, for example, apparently do not tolerate such methods of treatment. A great

[1] Approved for publication by the Director of the Wisconsin Agricultural Experiment Station and by the Division of Tobacco Investigations, United States Department of Agriculture.

variety of plant species has been used sufficiently to show adaptability over a wide range of hosts.

Strains of the variety of tobacco known as Havana-Seed were used mostly in the present experiments. For the virus tests, the hybrid host (*Nicotiana tabacum* × *N. glutinosa*) was used. This plant yields good local lesions within 3 days with the ordinary tobacco-mosaic virus.

The detached leaves were congested individually by applying high water pressure to the cut end of the leaf midrib or petiole in equipment as previously described (3). Each leaf usually required 5 to 30 minutes' treatment under water pressure to effect water congestion of one-half or more of the leaf area (Fig. 1, C, D). The congestion may disappear after 1 to 3 hours of drying or it may be maintained up to 24 hours in the containers without seriously affecting the subsequent condition of the leaf. The time that the water congestion is allowed to remain in the leaf may be regarded as the incubation period, although it may often overlap with both long penetration periods or short periods required for disease development.

The inoculum used in most of the trials was a beef-broth-peptone suspension of the tobacco wildfire organism [*Phytomonas* (*Bacterium*) *tabaci* (Wolf and Foster) Bergey *et al.*]. One advantage of using this organism was its ability to produce a typical toxin in culture, which could easily be studied separately by merely killing the bacteria in the culture by sterilization, leaving the toxin active. When a culture of the wildfire organism is used at high concentrations (below approximately 1 to 100), it may be difficult to distinguish between the effect of the toxin and that of the organism. The blackfire organism [*Phytomonas* (*Bacterium*) *angulata* (Fromme and Murray) Bergley *et al.*] provided a closely related bacterial parasite lacking appreciable amounts of toxin, although the wildfire organism can also be freed from the toxin by special methods.

Several fungi have been used in the trials including normal saprophytes such as *Aspergillus niger* v. Tiegh. and *Penicillium spp.* Other organisms such as *Septoria lycopersici* Speg. and *Helminthosporium sativum* Pam., King and Bakke not parasitic on tobacco, and some typically parasitic only on other organs of tobacco (*i.e.*, *Thielaviopsis basicola* (Berk. and Br.) Ferr., *Pythium sp.* and *Rhizoctonia sp.*) have been inoculated to tobacco leaves. Only one typical fungus parasite of tobacco leaves, namely, *Cercospora nicotianae* Ell. and Ev. was available for this study.

The virus used in the studies was that of ordinary tobacco mosaic which produces local lesions in 2 to 3 days on detached leaves of the hybrid host used when properly inoculated. The inert substances used most frequently have been India ink, which may be regarded as of a particulate size roughly comparable to bacteria, and water-soluble dyes such as rose bengal, which is of only molecular size as regards the orifices through which it will pass. Eosin and safranin have also been used with good results.

These various materials or inocula may be applied in various ways such as by droplets, immersion in baths, by wiping methods, or by atomizing with

varying forces, and in the presence or absence of different kinds of wounds or positions of stomatal openings. The inoculum may be largely or entirely removed from the leaf surface with running water at any desired time following application, thus in some measure regulating the period of penetration allowed. This has been usually less than 3 minutes (immediate), 1 to 3 hours (slow), and 18–24 hours (very slow). In the case of the organisms, a continued environment favorable for visible expressions of infection and disease were maintained in the refrigerator-pan moist chamber. A considerable part of the experiments dealt with the period that the water-congested leaves were allowed to remain congested following inoculation, but these results, although showing striking influences on the severity of disease, are not pertinent to penetration or original infection.

EXPERIMENTAL RESULTS

A considerable variety of combinations of conditions were usually compared in each individual experiment, which does not permit a convenient and detailed summarization of an extensive amount of data. For present purposes, it appears sufficient to present a summary of the typical results (Table 1). Since all the conditions of the individual trials, in some cases, cannot be constantly controlled (*i.e.*, stomatal position), exceptions to the results presented may be expected in efforts at close replication in some groupings. Considerable variation may be expected, especially between unwounded greenhouse-grown and outdoor-grown leaves. The location of the more irregular or variable behavior is indicated in the table.

Behavior of Normal Leaves

Considering the data largely from the point of view of water congestion in the leaf tissue and the possible avenues of penetration by various substances including different types of organisms, the results with normal (uncongested or control) leaves may be presented first. The normal or control leaves were in all cases tested simultaneously with the other forms of treatment and, except for such treatments, the conditions for penetration, infection, or disease development were approximately identical.

Reference to table 1 shows that none of the substances or organisms used appreciably penetrated uncongested leaves or caused infection. *Cercospora nicotianae* yielded some signs of infection in a small percentage of inoculations after prolonged incubation periods. If droplets of inoculum are allowed to remain on the leaf for extended periods, it is possible that minute amounts of water may enter the substomatal chambers, thus promoting penetration. Although water and dye evidently do not enter from surface droplets on uncongested leaves, the immersion of normal leaves occasionally favors congestion and penetration of substances, thus suggesting a very delicate balance in pressures. In any case, the infection tubes of fungi may presumably penetrate the stomata through their own efforts in the absence of water congestion, which the bacteria are evidently unable

to do. It is probable, however, that this form of fungus penetration is so slow and uncertain that infection is rare or, if it occurs, the injury may be small.

When a normal leaf is wounded, however, a new set of conditions arises and very different results are secured (Table 1). This is partly due to the removal of the epidermal barrier to penetration and partly to the congestion of the intercellular spaces adjacent to the wound. Even if surface water is not present on the wound, the cell sap released by wounding is often sufficient to cause congestion. When droplets of water-soluble, nontoxic dye or India ink are placed on needle-prick wounds, these

TABLE 1.—*Typical results on penetration and infection[a] of detached tobacco leaves treated in various ways with regard to type of water congestion and of wounding and subsequently maintained in moist chambers for several days*

Material or inoculum applied	No water congestion			Internal water congestion		Water congestion by atomizing	
	No wounds	Needle wounds	Wiping[b] wounds	No wounds	Needle wounds	Slight[c]	Distinct[d]
Dye (rose bengal)	0	3,7,4	2,7,4	2,8,4,9	3,8,4	2,4,9	3,8,4
India ink	0	3,7,4	1,7,4,9	1,8,4,9	3,8,4	1,4,9	3,8,4
Virus[e] (tobacco mosaic)	0	3,7,9	3,6,7	2,4,9	3,7,9	2,4,9	3,4,9
Toxin (from *B. tabacum*)	0	3,7,5	2,7,5	2,6,8,9	3,5,6,8	2,5,9	3,6,8
Bacteria (*B. angulatum*)	0	3,4	0	1,6,8,9	3,6,8	1,6,7,9	3,6,8
Fungus (*C. nicotianae*)	1,7,9	2,7,9	0	1,5,6,9	2,5,6,8	1,5,7	2,5,6,8
Fungus (*T. basicola*)	0	2,7,9	0	1,6,9	2,6,8	0	2,6,8

[a] 0, No penetration.
1, Very slow penetration (18–24 hrs.).
2, Slow penetration (1–3 hrs.).
3, Immediate penetration (1–3 min.).
4, No further progress.
5, Chlorosis.
6, Necrosis.
7, Small area or incipient infection.
8, Large area or heavily diseased.
9, Irregular behavior, depending upon stomatal openings, degree of wounding, etc.
[b] With cloth and carborundum.
[c] No visible permanent injury to cuticle.
[d] Some lasting injury to cuticle on uninoculated area.
[e] On hybrid host.

inert substances enter the congested area at once, as do bacteria or other particles of sufficiently small size to enter the intercellular spaces (Fig. 1, A). Large spores or mycelium may become only lodged in the wound itself, but infection tubes may subsequently enter the congested area if the condition is maintained for a sufficient time to permit their growth. The further progress of wound infections may naturally be checked quickly in leaves if the water supply in the area is lowered to normal amounts, or below, by evaporation. Frequently, little or no progress is made even by virulent parasites in wounded areas of otherwise uncongested leaves maintained under favorable environment for disease. In the instance of organisms producing toxins, chlorotic areas may be conspicuous in the infected area in the presence of very little necrosis.

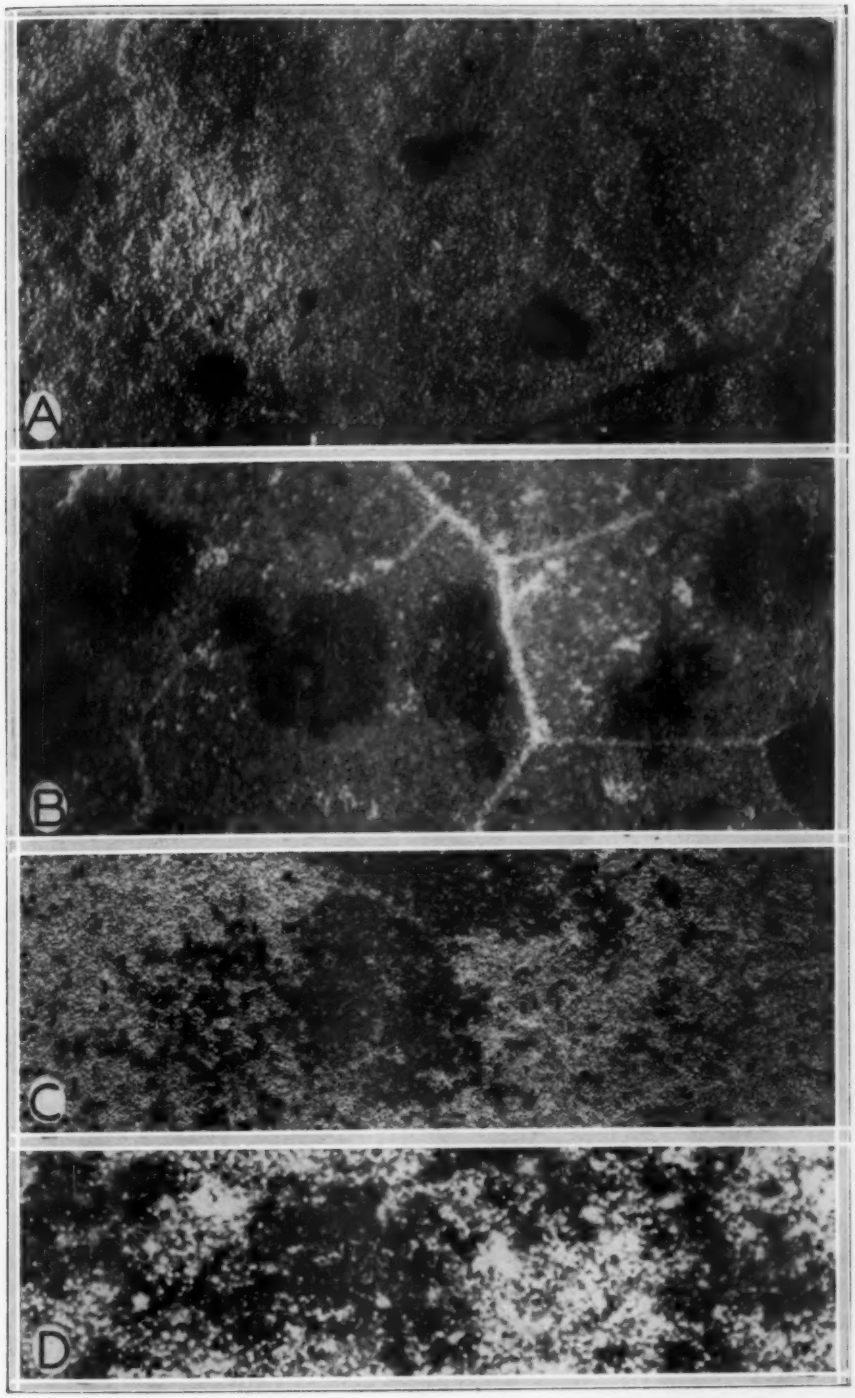

FIG. 1. Water-congested areas of tobacco leaf. A. As shown with India ink following wounding with tiny needle pricks (×7). B. India ink propelled into leaf by atomizing (×7). C. Water congestion induced by internal water pressures, photographed without staining (×7). D. An area similar to C greatly enlarged.

If the normal leaf is wounded by the "wiping" method, as used in plant virus inoculation, especially in the presence of an abrasive such as carborundum powder, only the plant hairs may be broken or the epidermal cuticle and cell walls scratched. Water congestion does not develop in intercellular spaces, and India ink or organisms do not enter into the intercellular spaces. However, particles of molecular size such as dyes, toxins, and viruses may enter cells through the broken leaf hairs or epidermal cell walls, resulting in coloration, toxic action, or virus infection, respectively. This phenomenon is rather remarkable and unique with the tobacco-mosaic virus, since water from external sources apparently is not necessary for penetration and infection to occur. Infection has been secured with this virus by wiping the leaf surface with dry and aged powder made from mosaic-infected leaves. It seems likely, because of immediate virus penetration into slightly wounded cells, that some force similar to that in water-congested, intercellular spaces is concerned in this form of invasion. Infection with bacteria and fungi is evidently not obtainable by the wiping method, although it is admittedly difficult to perform it without some injury which may permit invasion into intercellular spaces.

Behavior of Internally Water-congested Tissues

When detached leaves are artificially water congested by internal water pressure for experimental purposes, and inoculations made in an identical manner to that of the uncongested or normal leaves, more favorable conditions for penetration develop in the unwounded tissue and a highly intensified type of result occurs adjacent to the wounded tissues.

Penetration through the stomatal openings in the congested, unwounded leaves is of chief interest in relation to host predisposition and disease resistance. It is not certain that more stomata are open because of the water congestion, although it may be assumed that the stomata tend to open in the presence of excess water in the intercellular spaces. It is readily demonstrable, however, that dyes, India ink, and bacterial pathogens enter the intercellular spaces of congested leaves through stomatal openings whereas little or no such entrance occurs in uncongested leaf tissues. When the entire congested leaf is immersed in dye or ink, it is often apparent nevertheless that only a small fraction of the stomata are actually penetrated (Fig. 2, A and B). By the immersion method, too, it is obvious that the penetration is often localized in certain areas of the leaf, that it is influenced by the prior treatment or exposure of the leaf with respect to nutrition, environment, etc., also that it varies greatly with the plant species. Entrance of India ink through the stomata is relatively slow as compared to entrance through wounds; no doubt a consequence of the relatively small orifice through which the material must pass.

It is of interest to note that although the virus of tobacco mosaic can be shown to enter the intercellular spaces for considerable distances, no multiplication or disease results unless some cells are only slightly wounded

in some such way as to permit entrance into living cells. The wildfire toxin appears, on the other hand, to enter the unwounded cells from the intercellular spaces, resulting in gradual destruction of the chlorophyll and consequent chlorosis of the affected tissues. The action of the tobacco wildfire organism and certain other bacteria in the intercellular spaces is rapid in water-congested tissue, often producing severe necrosis and collapse of the tissue in 24–36 hours (Fig. 3, D). The signs of infection with fungi may be considerably slower in developing than with bacteria in congested leaf-tissue, possibly because invasion must be made by progressive mycelial growth rather than direct bodily penetration. Under some conditions with other

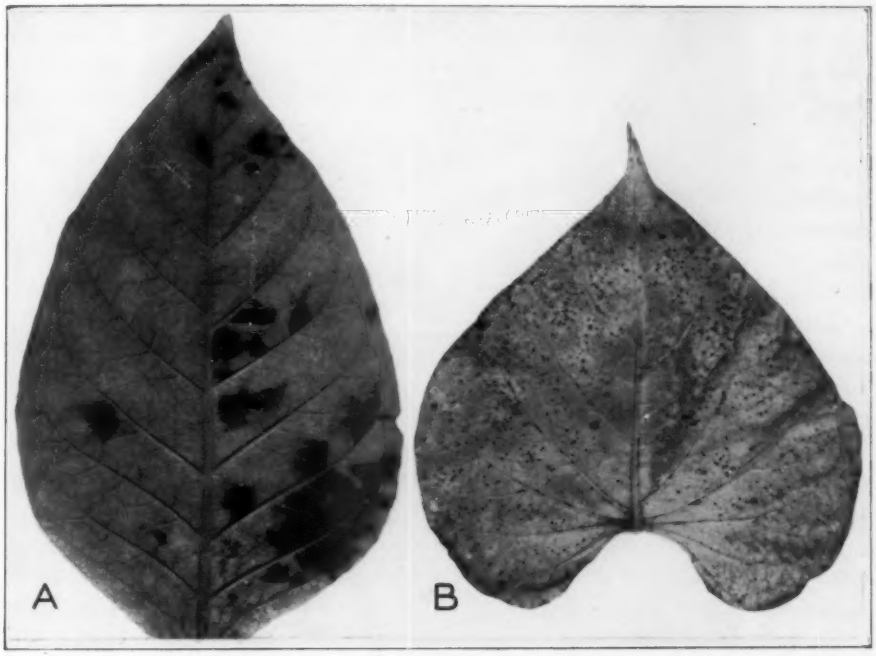

FIG. 2. Stomatal penetration in internally water-congested leaves. A. Tobacco immersed in India ink. B. Morning Glory immersed in rose bengal for about 5 minutes.

hosts and fungus parasites (*e.g., Helminthosporium sativum* on barley), symptoms of infection have been secured in congested leaves quite as rapidly as with bacterial parasites. With the fungus saprophytes used (*e.g., Aspergillus, Penicillium*), infection is slow and uncertain. However, *Thielaviopsis basicola*, typically parasitic in nature on roots only, makes surprisingly good progress in growth on water-congested tobacco leaves (Fig. 3, B). The endospores of this fungus are approximately the size of bacteria, suggesting that these may bodily penetrate the stomata and intercellular spaces before germination.

Infection with such leaf-parasitic fungi as *Helminthosporium sativum* and *Septoria lycopersici*, not known to occur on tobacco, has been more con-

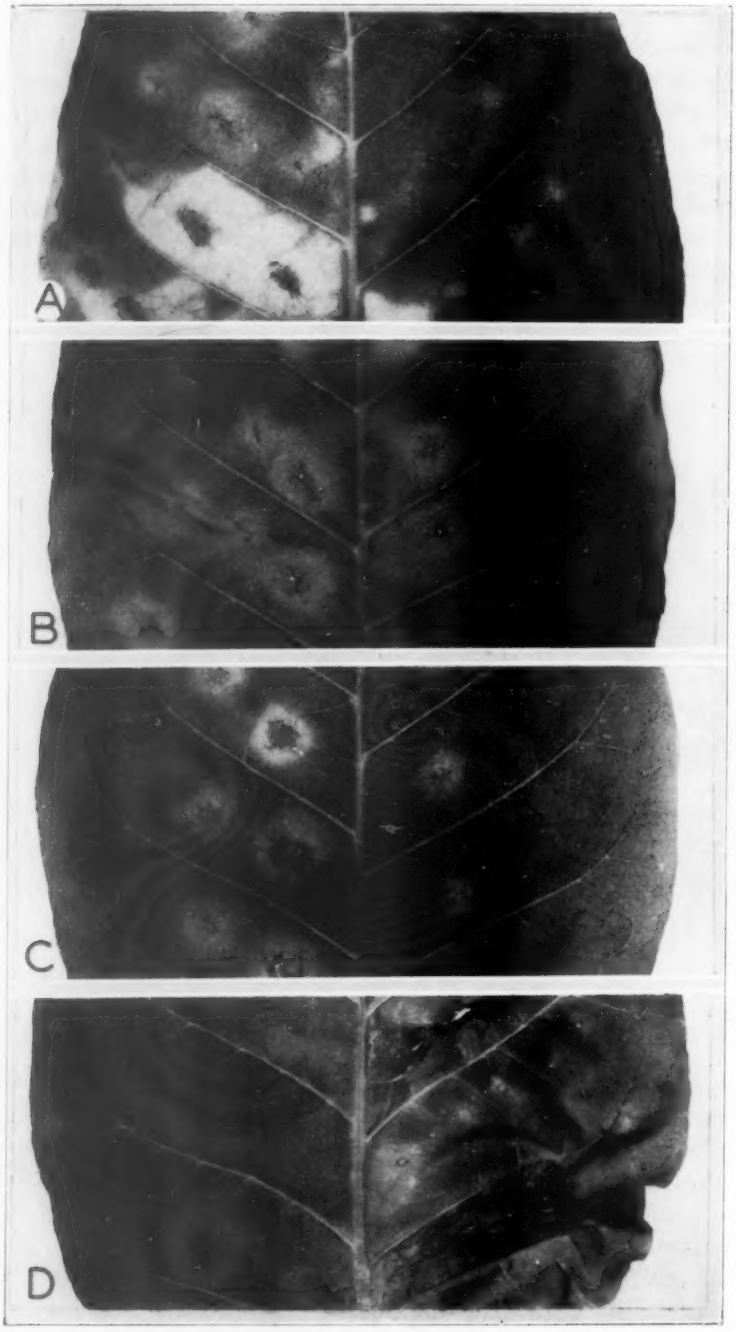

FIG. 3. A. Water-congested tobacco leaf, inoculated with *Cercospora nicotianae*. Wounded on left, not wounded on right side. B. Water-congested leaf inoculated with *Thielaviopsis basicola*. Needle scratches left side, needle pricks right side, unwounded far right (no infection). C. Localized water-congested areas produced by atomizing to varying degrees preceding inoculation with *Cercospora nicotianae;* far right, uncongested but inoculated (no symptoms). D. Portion of large internally water-congested leaf with different treatments applied between lateral veins. At lower left wounded and unwounded areas are shown and at lower right wound inoculations with a toxin-free and a toxin-containing wildfire culture are shown.

spicuous on congested leaves than that from *Rhizoctonia sp.* and *Pythium sp.*, which normally occur on tobacco as stem parasites.

In contrast to considerable variability in stomatal penetration of congested leaves, the penetration following wounding occurs with certainty and regularity (Fig. 3, A). The removal of the normally present barrier to penetration in the epidermal layers in previously water-congested areas produces an unusually favorable situation for both penetration and infection. The conditions are essentially the same as for stomatal penetration of congested leaves except that the relatively large orifice of the wound permits the surface water on the wound, together with suspended particles, to gush immediately into the surrounding intercellular spaces for distances up to a centimeter or more. This action places a parasite, when present, in a location comparable to that of a weak culture solution in a microscopic-sized moist chamber, thus providing for limited multiplication and growth, which in turn results in disease expression in the host tissues.

In wounded-congested tissue, special interest surrounds the force which causes water and suspended substances to be drawn into the intercellular spaces with such rapidity and consistency as to suggest a reduced pressure in the intercellular spaces. Actually this force can be overcome by a maintained positive water pressure in the leaf of approximately the magnitude of guttation pressure, but there is no evidence that the inward force is of a true physiological nature. The movement of water and suspensions through wounds is also too nearly instantaneous to be attributed to simple diffusion, but is more likely related to surface tensions, or other attractions which in turn develop quick-acting, capillary forces through the stomatal openings and the narrow channels of the intercellular spaces. Almost identical behavior can be shown by touching a capillary tube containing water to the surface of India ink, or by connecting drops of ink and water on a glass slide by a narrow channel of water. Entrance of solutions or suspensions into the channels of the intercellular area is not only immediate but it may continue for a considerable period under proper conditions, so that succeeding drops applied may often be observed to be slowly drawn into the orifice in the epidermis.

Congestion with External Water Pressures

In some respects there is little essential difference between internally produced water congestion and "storm water soaking" brought on by external water pressure (1). Both forms may fill large or small areas of intercellular spaces with water (Fig. 1, B) and, while part of the storm water soaking results from the forcible propulsion of water through stomata and through the wounds produced, this condition may induce further congestion by capillary force, as well, when surface water is present. The important difference in the two forms lies in the fact that storm-water-soaked areas are usually or probably wounded, thus most likely obscuring the part played by stomatal penetration and natural water congestion when occurring together.

In the present series of experiments, we have repeatedly produced various degrees of water soaking by atomizing the lower surface of detached leaves at a fairly constant water pressure for various lengths of time. Subsequent treatments with the various substances or inocula applied and other conditions of the test have been the same as for the controls and for leaves congested by internal water pressure. The degree of wounding, or effect on the cuticle and epidermis, may be often readily observed in such water-soaked, uninoculated areas, which eventually develop a blemish or "sheen" on the surface as frequently seen in field-grown crops following storms. The experimental irregularities and inconsistencies arise in efforts at producing water congestion of the external type with the minimum of injury to the cuticle. Leaves and parts of the same leaf vary greatly in the force required for the propulsion of water through stomatal openings, congestion occurring very easily in some instances without any visible evidence of surface injury. The best criterion yet found for determining the absence or presence of minute wounds in water-congested tissue is through the application of water-soluble dyes, such as rose bengal, to the treated area. If the dye enters immediately, wounds are present; if the dye enters slowly or not at all, the absence of wounds is indicated. When the atomizing is fairly heavy, yielding conspicuous, water-soaked areas with varying degrees of wounding of the epidermis, the results (Table 1), as might be expected, are almost identical with those of wounded, internally congested tissue (Fig. 3, B). The results from light atomizing were variable or irregular, probably because of the variation between no wounding and slight wounding. Although stomatal penetration may occur in such storm-water-soaked areas, there is naturally less certainty of the actual path of entry than in internally water-congested tissues (2). Also, there is no certainty that the natural protective cuticle or coatings on the leaf surface are not altered by any force of water sufficient to cause congestion when applied externally.

DISCUSSION

A series of investigations has been undertaken in this laboratory for the purpose of explaining the frequently observed erratic behavior of infection and disease. The predisposing influence of natural water congestion in the tissues has previously been emphasized (4). It was nevertheless clear that all water-congested tissues were not necessarily predisposed to infection, and that the occurrence of natural water congestion is a complex phenomenon involving many physiological factors in plants.

Infection studies on detached leaves, as reported in this paper, appeared to offer opportunities to test some of the more simple principles involved in infection. Substantially similar results have been secured on growing plants following natural water congestion. As the efforts progressed, however, it soon became evident that a wide variety of more detailed studies on growing plants should be performed and that the results with detached leaves were only of preliminary nature. Among these have been, for ex-

ample, the important differences that exist between outdoor and glasshouse-grown plants with respect to predisposition to infection. It has also been made increasingly obvious that leaves in a normal condition possess a remarkable degree of protection from penetration in their epidermal coatings, but that this may be greatly altered by nutrition and environment and often eliminated by wounding. When the epidermal barriers have been penetrated and the intercellular spaces invaded, infection is almost certain with normal parasites and is often likely with nontypical parasites and many saprophytes.

Of special interest is the relation of water congestion to physical penetration, the natural variability in species and varieties (3) to this phenomenon, and the behavior of stomatal or other natural openings with respect to their influence on the penetration of parasites into the water-congested intercellular spaces.

SUMMARY

The mechanism of infection in detached, artificially water-congested leaves of tobacco and other plants was studied in refrigerator pans used as moist chambers. A considerable number of different kinds of organisms were used including both parasitic and saprophytic bacteria and fungi as well as other substances such as toxins, viruses, India ink, and dyes.

The individual leaves were congested artificially by internal water pressure followed by the application of the inocula in various ways on wounded and unwounded surfaces, in comparison with adequate controls. The treated leaves were then maintained in approximately normal condition in the moist chambers up to 6 to 12 days if necessary.

Penetration did not occur through normal (*i.e.*, uncongested), unwounded tissues. Rare exceptions occurred with certain fungi such as *Cercospora nicotianae* but only after prolonged periods of incubation. Stomatal penetration frequently occurs through unwounded, water-congested tissues without the intervention of any injury to the cuticle or any exterior propulsion of the inoculum. Bodily penetration through the stomatal openings is dependent upon the presence of congestive water and leaf-surface water, with particles of sufficiently small size to pass through the openings. The mechanism involved is the establishment of a capillary force through the formation of a channel of water between leaf-surface water and the congestive water through which solutions or suspensions are transported.

Penetration in wounded plant tissues is usually immediate; and, when the leaf is also water congested, bacterial parasites in particular may be carried for considerable distances into the adjacent intercellular areas. In this location, the parasites find sufficient moisture and nutrients to multiply and hence cause disease in varying degrees depending in part upon the amount and period of water congestion in the tissues. Natural barriers to infection are destroyed by wounding and hence natural variations in resistance to disease may be impaired.

These observations permit a better understanding of modified predisposition of plants to infection, and of the natural varietal variation in resistance of plants to disease.

WISCONSIN AGRICULTURAL EXPERIMENT STATION
AND DIVISION OF TOBACCO INVESTIGATIONS,
UNITED STATES DEPARTMENT OF AGRICULTURE.

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PHYTOPATHOLOGICAL NOTES

The Relationship between American Tobacco Streak and Brazilian "Necrose Branca" or "Couve."—A virus disease resembling the American tobacco streak[1] was described from São Paulo, Brazil, under the names "necrose branca" (white necrosis) and "couve" (cabbage-leaved tobacco).[2] It was pointed out that the necrotic symptoms of this disease and the physical properties of the causal virus were similar to those of the American disease, but unlike tobacco streak, the necrotic symptoms were followed by incomplete recovery, and at this stage the affected plants showed leaf modifications (such as stiffness, narrowing, petiolated leaves in the case of sessile varieties, etc.), flowers sometimes with petals partly separated and the constant presence of a filament-like appendage at the extremity of each petal.

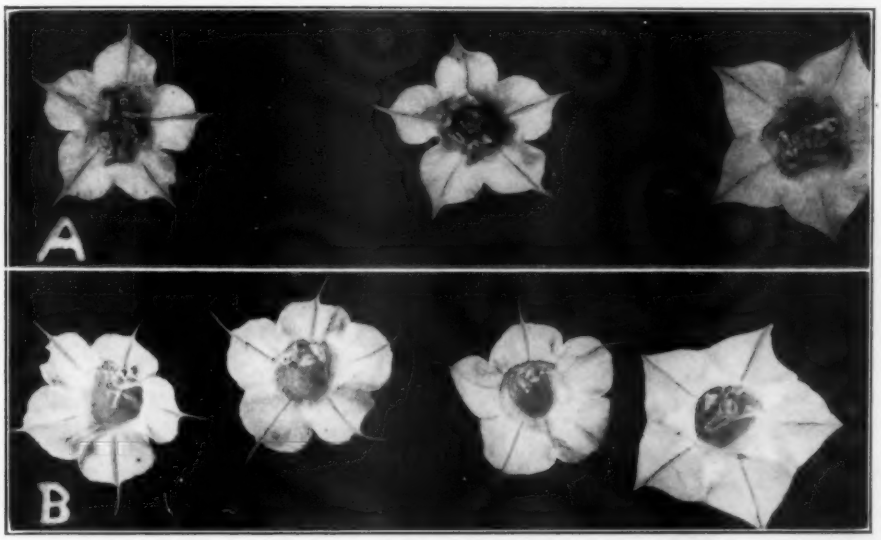

FIG. 1. A. Flowers from a plant affected by tobacco streak. Normal flower on right. Variety Turkish. (Photograph by J. A. Carlile.) B. Flowers from a plant affected by the Brazilian disease. Normal flower on right. Variety Bright Virginia.

During the writer's stay at the Rockefeller Institute for Medical Research, Princeton, N. J., in 1943, an attempt was made to prepare an antiserum to tobacco streak virus; this antiserum was to be taken to Brazil in order to test the serological relationship between the American and the Brazilian viruses, as the introduction of foreign diseased plant material for comparative studies was deemed inadvisable. Unfortunately, no active antiserum was produced and the relationship of the two viruses could not be tested by this method.

In the course of these studies it was noticed that plants of the Turkish

[1] Johnson, J. Tobacco streak, a virus disease. Phytopath. 26: 285–292. 1936.

[2] Costa, A. S., A. R. Lima, and R. Forster. Necrose branca—uma moléstia de virus do fumo (*Nicotiana tabacum* L.) e "Fumo couve" como sintoma tardio. Jornal de Agronomia 3: 1–26. 1940.

variety affected by tobacco streak had one characteristic feature of the Brazilian disease, hitherto not described in connection with the former: petals ending in a filament-like appendage. Figures 1, A, and 1, B, show this interesting symptom which furnishes additional evidence of relationship between the American and Brazilian viruses.—A. S. COSTA, Department of Genetics, Instituto Agronômico de Campinas, São Paulo, Brazil.

Agar Medium and Technique for Isolating Pythium Free of Bacteria.—Isolation of *Pythium* from guayule seedlings (*Parthenium argentatum* Gray) affected with damping-off and seedling root rot by usual laboratory methods was generally unsatisfactory. The isolates when obtained in culture were usually contaminated with bacteria and the common practice of acidifying the agar medium to separate the fungus from the bacteria was never too satisfactory. In the course of investigational work on damping-off of guayule, a special agar medium and a technique was developed which facilitated the isolation of bacteria-free *Pythium* from diseased plants and from the soil. The method was particularly effective in isolating *Pythium ultimum* Trow., which has been reported[1] as causing damping-off and a root rot of guayule.

This method has the advantage of yielding isolates of *Pythium* free of bacterial contamination from nonsurface-sterilized material. It also provides a simple and useful method of separating bacteria from *Pythium* and other fungi in bacterial contaminated cultures.

The formula for the agar medium is as follows:

Dextrose	10 g.
Ammonium acid phosphate ($NH_4H_2PO_4$)	2 g.
Potassium nitrate (KNO_3)	1 g.
Magnesium sulphate ($MgSO_4$)	1 g.
Agar	25 g.
Water, distilled	1000 cc.

The agar and dextrose were dissolved in 800 cc. of water by heating in a double boiler or autoclave. The other materials were dissolved in 200 cc. of water and added to the thoroughly dissolved agar and dextrose and autoclaved for 15 minutes at 15 pounds pressure. The usual procedure was to autoclave 1000 cc. of medium in three 500-cc. Erlenmeyer flasks. Each flask contained approximately 333 cc. of medium, and immediately after autoclaving the hot liquid medium was poured into sterile Petri dishes. However, the sterilized agar medium was occasionally stored in a refrigerator for several days or weeks. Liquefying the medium a second time by heating in a water bath did not adversely affect its qualities. The agar medium had a reaction of from pH 5.0 to 5.5.

The solidified agar medium, which was about 5 mm. thick in Petri dishes (9 cm. in diameter), was divided into 4 equal quarters with a small sterilized

[1] Campbell, W. A., and Bailey Sleeth. A root rot of guayule caused by *Pythium ultimum*. Phytopath. 35: 636–639. 1945.

metal spatula or wide scalpel. A small bit of inoculum consisting of diseased plant tissue or soil was placed in the center of each quarter section. Sometimes, as a matter of convenience, the inoculum was placed on the agar before the agar was quartered. Three of the sections were carefully lifted out of the Petri dish by means of a small sterilized flexible metal spatula and placed inverted in separate sterile Petri dishes. This part of the process was similar to that used in turning a pancake. The 4th or remaining section was inverted in the original Petri dish. A certain amount of care had to be exercised in inverting and placing the inoculated section of agar medium so that the inoculum would be completely covered with the agar block. An effort was made to create a seal around the inoculum by having the edges of the agar block come in contact with the bottom of the Petri dish.

If *Pythium* were present in the original inoculum, scattered hyphae grew through the agar block and reached the upper surface within 24 hours at laboratory temperature. If other slower growing fungi were present in the plated inoculum, hyphae of *Pythium* were the first to appear on the surface so that hyphal tip transfers usually resulted in pure cultures. When reisolations were made from infected seedlings in pathogenicity tests, it was not unusual to obtain bacteria-free *Pythium* in over 90 per cent of the attempts. The growth of bacteria was inhibited while *Pythium* and other fungi grew well on the special agar medium. Cultures of *Pythium* contaminated with bacteria were readily freed of bacteria by the same method.

In order to secure the best results, the agar sections should be fairly thick and the agar medium of sufficient hardness to permit lifting the sections without breaking. Since the quality of agar-agar varies, the amount in the formula may need to be varied accordingly in order to obtain the desired consistency of the agar medium.—BAILEY SLEETH, Special Guayule Research Project, Bureau of Plant Industry, Soils and Agricultural Engineering, United States Department of Agriculture, Salinas, California.

ANNOUNCEMENT

AMERICAN PHYTOPATHOLOGICAL SOCIETY

Annual Meeting

The American Phytopathological Society will meet with the American Association for the Advancement of Science in St. Louis, Missouri, March 27–30, 1946. The headquarters hotel for the Society will be announced in the January issue of Phytopathology.

Members wishing to read papers or to give demonstrations should submit triplicate abstracts, not exceeding two hundred words, to the Office of the Secretary before January 25, 1946. The time required for reading papers, not to exceed fifteen minutes, projection apparatus needed, and section preference should accompany the abstract. Abstracts of demonstrations should state the floor and wall space required as well as the electrical and other equipment needed.

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Junior authorship indicated by pages in "( )"

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Page 143, line 1, *read* Tilletia tumefaciens was founded by Sydow (See 5) *for* Tilletia tumefaciens Sydow

Page 145, *add* to literature cited, 5. Sydow, H. and P., and E. J. Butler. Fungi Indiae Orientalis. Pars IV. Ann. Mycol. Berl. **10**: 244–280. 1912

ERRATA, VOLUME 35

Number 2, Table of Contents, *read* H. R. Rosen *for* H. Rosen

Page 119, item 9 in literature cited, *read* Root defects and fungi *for* Root defects

Page 129, line 4, *read* presumably *for* presumbaly

Page 187, line 5, *read* Romania *for* Roumania

Page 239, item 3 in literature cited, *read* 1919 *for* 1939

Page 315, item 7 in literature cited, *read* 1945 *for* 1944

Page 340, line 41, *read* Tong-kin *for* Tungkin

Page 341, line 14, *read* D. C. Edwards *for* E. J. Edwards

Page 545, line 16, *read* In 1941 the percentage *for* In 1939 the percentage

Page 558, line 27 and Table 3, Col. 4, line 2, *read* 26.9 *for* 19.7

Page 743, line 2, *read* Pellicularia *for* Pellicullaria

Page 767, line 17, *read* X217 *for* X216

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